CHEMICALS IN FOOD
and in Farm Produce: Their Harmful Effects

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by

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To

IDDER

who

While Cherishing

Harries No Man's Cattle

The United States Fish and Wildlife Service of the Patuxent Research Refuge, Laurel, Maryland have taken great trouble in sending to me many books and journals not available in England. I am most grateful for this help which no one else could have given.

This book has depended on the assistance of Mr. P. Wade, Librarian of the Royal Society of Medicine, and of Miss Mary Evans, his deputy, who have always succeeded in finding for me the rarest journals. Their assistants by their kindness and their efficiency have made working in the library easy and a pleasure.

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CHAPTER I

Introduction

The deliberate or accidental addition of possibly injurious chemicals, of 'non-nutritive substances', to food is not new, but only in the last few years have ch additions become so universal that it is now virtually npossible not to eat several every day. The chemicals comonly present in food number many hundreds, and each ar new ones are introduced with an ever increasing emical complexity and with an unknown effect on the ody. The importance of knowing what these chemicals are nd how they may react on health is obvious. But the subct overlaps with so many disciplines—medical, nutrinal, agricultural, commercial—that it tends to be nored by each It is, indeed, almost a new and separate ience.

Food is eaten several times every day throughout our life. is, indeed, the very stuff of which we are made. Our health nd the future health of our children is wholly dependent on it. The individual himself is the only person who can, at least a great extent, control whether or no he and his family t safe or injurious food. It is puzzling that people think so uch about atomic warfare and so little about food, when ther may equally destroy them individually and the one roblem is beyond their control, the other theirs to do with s they like.

So this book is written for those who want information about this personal problem they themselves can solve. It considers what toxic substances may be taken in our foods: particularly those substances which do not cause acute though transitory illnesses like diarrhoea, but rather those which, insidiously gnawing away the body, may only tardily reveal their presence by causing or precipitating goitre, coronary thrombosis, disseminated sclerosis, cancer, etc.

Much time and vitality are spent by food reformers in protesting about the chemicals added to food. While the general principle behind such protests is wise, too often the particular abuses cited are either relatively harmless or the scientific evidence against them is buried in the highly specialized journals which few doctors and no laymen read. It is hoped that this book will not only help those who desire safer food to see where their zeal for reform is most needed, but will also help to provide reformers with scientific backing for their beliefs: beliefs which, though correct, are often purely intuitive or intellectual and so fail to impress those who rightly demand scientific evidence.

The references throughout the text refer to the bibliography at the end: they are given so that those who doubt may check for themselves evidence which is often startling, while those who are interested can amplify what they have read. Reading many of the journals, such as the *Acta* of the International Union Against Cancer, is more exciting than any novel or murder story. Even though many papers have a highly scientific background, yet the purport of these can generally be easily gathered without any knowledge of science.

The lay public are essentially the people who must decide for themselves and for the country as a whole what degree of safety in food they require and therefore what controls

Parliament may and must enforce, so scientific and medical terms have not been used or are defined as accurately as necessary to give the gist of any discussion.

Doctors, it is hoped, will find a book written in lay language, but corseted with scientific references, of interest when it deals with a subject which is scattered over many diverse journals and is never dealt with as an entity in any textbook or single journal. It is a subject which has many facets of interest in day to day medicine and is also a subject where the general practitioner is in a unique position to recognize and reveal what particular foods and diseases are related to each other: why a group of children suddenly develop goitre; why a young man of twenty-three has inoperable cancer of the stomach; why there is an outbreak of *herpes zoster; why an utterly normal man develops paralysis* agitans; or why someone is unreasonably tired.

Bacterial food infections and also allergy are not discussed. Both subjects are well known to the public: in fact 'clean food' in the sense of food free from pathogenic bacteria is too well known, since it has distracted attention from the more important problem of food which contains poisonous substances unrelated to germs. The Food and Drugs Amendment Act of 1954 was castrated because so much time was wasted in debate on the facile idea of cleanliness that little time or thought was spent on the difficult idea of the control of 'non-nutritive additions to food'. As a result any substances whatsoever—apart from a few classes of food additives like colours and preservatives—may be added to food unless specifically prohibited, in contrast to the food laws of many countries where nothing may be added to food unless it is specifically permitted.

Noxious substances may be present in food for four reasons:

Natural occurrence in foods which are generally per-

fectly wholesome. For instance cows fed on too many cabbages give milk which causes goitre.

Accidental contamination, as when at the beginning of this century some 6,000 people in the north of England were poisoned by arsenic in their beer.

Deliberate additions to food of substances of unknown but possible toxicity. This is the most worrying subject dealt with. It concerns, essentially, the artificial colours; the chemicals used in processing food, such as the agene of contentious memory used in flour; the emulsifiers, etc., used to alter the physical palatability of foods.

Residues of agricultural poisons which, used on farms to destroy insect pests, weeds, etc., may remain in eggs, milk, vegetables, grain, etc. The presence, for instance, of the insecticide DDT in human fat is due to cows licking the fly-killing DDT off the sprayed walls of their byres, then excreting it in their milk and so handing it on to people.

These four ways in which food may be rendered harmful are discussed in the first chapters while those illnesses which may be caused or aggravated by chemicals in food are considered later. The last chapter is on the destruction of wild animals, birds, butterflies, fish, etc., through the largely avoidable poisoning of their food supplies by agricultural insecticides, etc. The public is unaware of this destruction but, realizing it, may feel it is too high a price to pay aesthetically for a doubtful agricultural profit.

CHAPTER II

Naturally Occurring Poisons

In England it is extremely rare for common foo
come either obviously or insidiously poisonou
subject is important not only because of th
occasional tragedy which follows eating apparent
food but also because the covert toxicity of, for
some milk emphasizes the problems which can a
deviating from the customary habits of agricultur
traditional ideas of wholesome food.

Human milk may contain toxic substances eat
mother. The puzzling diarrhoea of some babies
due to their mothers taking laxatives, especial
'natural vegetable type', which often include senna
or colocynth. Or, again, breast feeding may appe
agree with the infant because its mother is ea
stewed plums, rhubarb or boiled new potatoes (
are sometimes startlingly sensitive to borax, s
mothers should avoid all dusting powders, nasal
douches, mouth washes, etc., from which they mig
and then transmit to their milk borax or boric acid.
lead poisoning, too slight seriously to affect the mo
be highly toxic to the suckling (47, 200);[1] nitrates i
water, though harmless to the mother, can re
dangerous (p. 31); and presumably some insectic

[1] Numbers refer to numbered items in Bibliograph
'References'.

by the mother in contaminated foods (p. 88) or inhaled from fly-sprays, etc., would be excreted in milk and could be highly injurious (p. 87). Many other chemicals may be excreted in the milk but for all practical purposes they present no problem, nor is alcohol harmful when confined to ordinary social events.

Cow's milk is worrying for two reasons: it may contain antibiotics, pesticides, etc., used in agriculture (pp. 71–87); it may cause goitre or, very rarely, acute illnesses because of toxic substances in the feed.

Simple goitre, that is a symptomless if unsightly swelling of the thyroid gland in the front of the neck, is in most parts of the world caused by a deficiency of iodine in the diet. The thyroid gland enlarges with the effort of making its iodine-containing hormone from meagre supplies of iodine. So such goitres, all over the world, have been prevented by the simple addition of iodine to table salt, by iodine tablets, etc., and this has become the routine and virtually infallible way of preventing goitre in countries where it is endemic. But, very annoyingly, in some areas, such as West Penwith in Cornwall, goitre is common in spite of ample iodine in the food, so there must be some other factor, apart from lack of iodine, which hinders the thyroid from making its hormone, and so leads to its enlargement to overcome this hindrance. When rabbits, in 1928, were found to develop goitre if fed predominantly on cabbage, it seemed possibly that a high consumption of certain vegetables—especially *Brassicae* and *Cruciferae*—might explain goitres on diets containing ample iodine. Reports soon appeared that monks, Indian villagers and sheep in winter all develop goitre when living largely on cabbages, swedes, turnips, etc. But the subject remained largely academic, as far as ordinary people are concerned, for many years until the very brilliant research by Clements and Wishart (2) in Tasmania in 1955.

When goitre was found to be endemic amongst Tasmanian children in 1949, iodine tablets were distributed and abolition of goitre was confidently awaited. But after five years the number of goitrous children had, in some areas, not decreased but greatly increased. The explanation of this startling finding was ultimately traced to the children drinking more milk, due to the introduction in 1950 of a free milk scheme for children. To provide the extra milk required, the farmers had fed their cows on marrow-stemmed kale (*Brassica olenacea moellerii*). The goitre-producing substance in this kale had been excreted in the milk and had then affected the children.

Since kale, rape, turnips, etc., are largely fed to cows in England and since even pastures contaminated with cruciferous weeds may cause milk to be goitrogenic, it would seem important to know how goitrogenic English milk is now, and how goitrogenic it may become with increasing reliance during the winter on home produced fodder crops like kale. Further, it has been suggested that the goitre caused by vegetables differs radically from that due to simple lack of iodine: the latter is abolished by iodine and the thyroid gland becomes normal, while the former is not benefited by iodine but the gland may instead in later life be stimulated to become toxic. If this is true, the Government may be wise in letting lapse its decision to add iodine to our salt, at least until more is known about what proportion of English goitres are due to lack of iodine. There is, indeed, already slight evidence that the Londoner's food is goitrogenic (3).

Boiling Brussels sprouts, cabbages, turnips, etc., does not destroy their goitrogenicity (19), but so many generations of children have been forced to eat so many, so very many cabbages, that the goitrogenic effects of vegetables in our diet must be negligible or we should all be goitrous.

The milk of cows which have been fed on poisonous ivy or

the leaves of the common artichoke, is said to cause diarrhoea and vomiting, while infants have been poisoned by the milk of goats grazing on *Colchicum autumnale* (4). Milk from cows fed on potato mash from a distillery has poisoned children, possibly because it contained solanine (p. 28). passing, Abraham Lincoln's mother should be remembere having drunk the milk of a cow which had fed on whit snake root, she died: so did many other settlers from suc milk or from its butter and cheese.

The penicillin in the milk of cows, treated for mastitis w this antibiotic, may evoke allergic symptoms (p. 72).

Honey poisoning was accurately described by Xenophon i 400 B.C. when, after leading the ten thousand Greeks bacl from Persia and the walls of Babylon, they at last came to the yearned-for sea near Trebizond. 'The Hellenes marched up the mountains and encamped in a number of villages with plenty of provisions. They found nothing remarkable there except the great number of beehives; all those who ate the honey went out of their senses and vomited and purged and not a man of them could stand straight on his feet. If they ate only a little they seemed like drunken men, if they ate much, like madmen; some even died of it. So they lay in heaps as if there had been a rout, and they were very unhappy about it. Next day no one died, but about the same time of day they came back to their senses; in another day or two they got up dazed as if they had been drugged' (5). Pulewka (6) from Turkey, where poisoning is still common, has given a full review of the literature. The poison is andromedotoxin, which is in the nectar of rhododendrons—especially *Andromeda*—azaleas, oleanders and the dwarf laurel *Kalmia latifolia* (p. 22). Treatment, only safe in the hands of a doctor, is to wash out the stomach with potassium permanganate.

In England honey poisoning has not been described, but

arms and neck, and, always, weakness of the legs, so that in severe cases the patient cannot stand or, in milder cases, staggers and reels as if drunk. The mouth and throat feel hot; speech and swallowing become more and more difficult; ultimately death occurs from paralysis of the respiratory muscles, though breathing appears normal until near the end. The mind generally remains clear but an odd feeling of lightness, as if the patient could fly, is common. Pain in the bladder is usual but vomiting or diarrhoea are rare. The pulse is feeble, but the reflexes and the temperature remain normal. Nothing definite is found P.M.

Treatment in essence is to regard the condition as an acute emergency: admission to a hospital with a respirator must be arranged at the first suspicion: while waiting the patient must be made to vomit: he must not have alcohol: ordinary manual artificial respiration has been known to keep a man alive until his paralysis waned. If he lives 12 hours the outlook is good.

The poison—mytilotoxin—in the mussels, or at least from similar mussels from the Pacific coast of North America, is derived by them from eating a plankton of the genus *Gonyaulax* (14).

Lockjaw or tetanus has been treated in the last few years with derivatives of curare or 'arrow poison'. This method of relieving muscle spasms and convulsions with a paralysing poison was acclaimed, rightly, as a great advance in treatment. So it is good for pride to read what Dr. Crump of Dublin (15) wrote in 1872. He was investigating some mussels which had paralysed and killed three children, when a woman of fifty came under his care with severe tetanus from a wound on her leg: 'Jane Cantillon had a fine set of teeth; with difficulty and manœuvring I contrived to slip the handle of an iron spoon on the flat between her molar teeth. . . . I so far wrenched her jaws asunder that I could introduce

my forefinger into her mouth. I gradually now introduced into her mouth one of these unboiled mussels and with my forefinger pushed it down into her pharynx so that she was compelled to swallow it.' She was given two more mussels and in three hours 'she had a placid natural countenance—could open her jaws to the fullest extent and also move her neck freely. She had more use of her hands, but her lower limbs remained rigidly stiff.' She recovered completely.

Oyster poisoning similar to mussel poisoning occurs in Japan but probably never in England, only one doubtful case being reported (16).

Fish poisoning is mentioned vaguely in many books which fail to differentiate between fish caught in English seas or rivers and fish caught in the West Indies and Japan (10, 17) where, indeed, fish may be fantastically toxic. Thus the celebrated conger eel which paralysed five people and writhes through many books was not a modern English conger eel, as is generally implied, but was caught and eaten 150 years ago off the West Indies (10).

Indeed we in England are only left with the faint possibility that the roes of some freshwater fish are toxic, and this but rests on gossip and, for the spawn of barbel, on Izaak Walton: 'It is dangerous meat and especially in the month of May. The learned Gesner says it may even endanger life.'

But there are two foreign diseases caused by eating fish which have not yet appeared in England, though there appears no reason for our continued immunity.

Haff disease (236) has occurred, both in East Prussia and Sweden, about 18 hours after eating cooked fresh water fish, and especially their livers. There is an explosively acute paralysing tenderness of the muscles, followed by myoglobinuria which turns the urine brown or black. Recovery takes 12 to 48 hours unless death is caused through the

Oxalic acid is also found in many vegetables and fruits such as *spinach*, *sorrel* and *strawberries*: here it is never in such large amounts that it is dangerous though it may in sensitive people cause oxaluria—crystals of calcium oxalate in the urine. These may scrape the urinary tract, causing acute lumbar and abdominal pain and a painful, bloody urine. I have found that such patients can often safely eat strawberries if they also take with them very large amounts of cream: an interesting vindication of the traditional custom of adding cream to all foods rich in oxalic acid.

Lettuce poisoning I first heard of many years ago when a young friend, doing his first country locums in the West of England, found two unconscious people in a cottage. While still puzzling why and what to do, the district nurse happened to arrive and contemptuously told him they had, as many people did, eaten stewed bolted lettuce which, of course, sent them to sleep. I have never heard of this again, though some people find ordinary lettuce leaves make them sleepy; but in the last century Lauder Brunton (22) was using lettuce opium 'to allay cough, quiet nervousness and induce sleep in cases where, from idiosyncrasy, opium is not borne'. In the eighteenth century Coxe (23), trying to make America independent of foreign opium, reviewed earlier authors on the value of lettuce including Dioscorides, and he himself found the dried juice therapeutically just like opium when it was collected properly—it must be allowed to flow out by itself, without squeezing, from superficial circular cuts made in the stalks when seeding is starting. Dried juice used to be extensively exported from Scotland, France, Germany, and, though it was costly, from Russia.

Potatoes normally contain very small amounts of the poisonous alkaloidal glucoside solanine, which is increased in unripe tubers; in those lying on the surface of the ground and so becoming green; in those grown with much potash;

and in those which have begun to sprout. Salaman, in his classical book (24), states that there is no evidence that solanine has ever caused trouble, but reading accounts of potato poisoning forces one to conclude that this is a very real condition which makes essential the domestic waste of all potatoes if they are green or sprouting. Thus in 1917 in Glasgow, 61 people in 18 households were ill after eating potatoes, all from the same consignment to a Co-operative Society (25). Many of the potatoes were sprouting, when examined later, and those from the only household where one patient died had about ten times the normal amount of solanine. The symptoms (25, 26), which may come on within two hours, are generally headache and vomiting and diarrhoea; and sometimes debility, weakness, drowsiness, apathy, jaundice, partial paralysis and fever. It is said that children in France have been poisoned by the milk of cows fed on potato pulp from distilleries (26), and in Germany the cows themselves have died (25).

Nutmeg (277) is dangerous in large amounts: even one, pounded up as a medicine or as an abortifacient, causes numbness, dizziness, thirst, headache, swelling of the face, abdominal pain and delirium. Recovery is usual, but children have died.

Broad beans (*Vicia fava* or *Vicia faba*) cause a peculiar illness known as Favism or Fabism which is most probably allergic. For many years it was thought to be confined to people of Mediterranean origin, but recently a 9-years-old boy, whose parents and grandparents were English, developed the condition (29) For three years running, after eating the home-grown broad beans of his grandmother he had attacks of shivering, malaise, vomiting and jaundice. His maternal great-uncle had had similar attacks after eating broad beans. The condition may be trivial or fatal and is, in essence, a haemolytic anaemia. Symptoms come on 5 to 24

feeds made up with nitrate-containing water, they may develop within a few days a greyish-blue or brownish-blue cyanosis round the lips, which rapidly spreads to the fingers and toes, face and whole body. There may also be some gastro-intestinal upset.

Such 'blue water' babies must be admitted to hospital urgently, since death may occur within 36 hours of the cyanosis first being noticed. Giving them only boiled water —the classical domestic treatment for infantile gastro-enteritis—may be fatal, since the nitrates, far from being destroyed by boiling, are concentrated. This cause of 'blue' babies has only been recently recognized, but already four cases, one of them fatal, have been reported from Suffolk and Worcestershire. Typical of these is the girl of five weeks (30) who, after one week on artificial feeds made from water got from a well close to her caravan, became cyanosed, though her mother and two older children drinking the same water were not affected. She completely recovered in hospital but relapsed on returning to the caravan because, though the original well was not used, the one which was used instead also gave nitrate-containing water. (The problem was solved by the father bringing home water every day from the town where he worked.) It is even said that infants suckled by women drinking such waters may be affected.

Domestic detergents (61), surprisingly enough, now commonly pollute our domestic water supplies. This is because about half the harmless domestic soap previously used is now replaced by detergents which once they are dissolved in water cannot be got out of it again in any feasible manner: thrown down the sink in the washing-up or laundry water they fail to be destroyed, as soap and most of the other ingredients of sewage are, at the sewage farms and so flow out with the purified sewage effluent into rivers. This polluted river water is the source of much of our drinking water.

Detergents consist essentially of various mixtures of 'surface-active' agents, mixed with ancillary substances known as 'builders' and also, sometimes, with colourless dyes used to give a brightness to clothes. The composition of the 'builders' is not clearly known and doubtless varies and will vary at the whim of the makers: so injurious substances may or may not be present. One would be happier if the complete composition of detergents were given on the outside of the packet. One would be even happier if stilbenes (p. 149) were not the basis of some detergents' colourless colour (62), since some stilbenes are highly cancer-producing, at least for animals (p. 62). The surface-active agents themselves—their chemical comp[...] —are not li[...] severa[...]) [...] occurrence of serious effects [...] much longer term cannot be ignored . . . and since it is not possible to argue with certainty from animal experiments to man, the possibility will need to be kept under careful review' (61). The real worry, however, is not the toxicity of the 'surface-active' agents themselves but the risk that they may so alter the physical conditions in the intestines that injurious substances which normally cannot pass through the intestinal wall into the body are enabled to do so (62).

Swallowing detergents is now almost unavoidable several times a day all the days of our life. About 2 milligrams a day will be taken in food and drink prepared in utensils which have not been carefully rinsed after being washed with detergents, and to this must be added as much as another 3 milligrams from our tap water during dry weather. The Hertfordshire Colne, for instance, at a time when the river's flow was good, and so was diluting the sewage effluents, contained nearly 5 milligrams of 'surface-active' agents per litre.

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children, especially, sugar in the urine is a common finding.

But beer, though it dissolves lead less well than does cider, is the classical cause of lead poisoning: beer remains overnight in lead pipes leading from the beer barrels to the bar, and so dissolves large amounts of lead. Such beer, either given to the regular early morning customers or returned to the barrel (44), has caused many cases of poisoning. Lead pipes in the past have also been used for ordinary water supplies which, especially when very soft, may dissolve so much lead that an epidemic of abortions has been caused (44). Though in passing it should be pointed out that lead used as an abortifacient generally kills the mother and not the foetus: one vaguely ill pregnant girl in a hospital ward was recently regularly poisoned with lead at each visit of her most solicitous young man, but she did not abort, her condition being at last correctly diagnosed as she became comatose.

Domestic poisoning is most likely to occur from the use of earthenware cooking pots lined with a cheap lead glaze which dissolves with acid fluids: two women were acutely ill after stewing cranberries in such a pot (4) and thirty people suffered when similar pans were used in the brewing of home-made beer and wine (45). At Brest 37 people were poisoned by bread made in ovens heated by lead-painted wood from old boats (46), though it is fear of spreading bugs rather than of lead poisoning which makes some *départements* prohibit the use of *bois de démolition* for breadmaking. Among other ways in which lead poisoning may surreptitiously appear in the highly sensitive infant and child are the use of lead nipple shields during suckling (47); the transference, it is said by French authors (47), of lead to the foetus and suckling by mothers who have used cosmetics and hair dyes containing lead; the gnawing of lead soldiers or paint off cots, play-pens and toys; the chewing of

yellow or red crayons coloured with lead chromate; the licking up of rain drops as they run down lead-painted verandas after a spell of hot weather; the eating of flakes of peeling paint; the eating of apple peel (p. 98). About 4 children yearly die from lead poisoning and many more must be poisoned but never diagnosed. Dogs may be affected by lead, the symptoms simulating distemper or canine hysteria (146).

Copper poisoning from copper pans, etc., has long been feared by housewives, not without cause. 'On Monday, August 25, 1783 a young lady, 17 years of age, amused herself, whilst under the hands of the hairdresser, with eating pickled samphire of which she consumed two breakfast plates full, amounting to three to four ounces.' She then slaked her thirst with vinegar and died after ten days of incessant vomiting Percival (49) found the pickle was heavily impregnated with copper; he also gives accounts of other severe cases of copper poisoning and then urges that iron cooking pots should replace the copper ones used in the navy, since he believed these caused the desolating scurvy which brought so many voyages to disaster—an interesting observation, since it is now known that the minutest trace of copper during cooking destroys vitamin C, lack of which causes scurvy. Ross (50) has given an excellent review of current copper poisoning, including 9 cases of his own caused by apples being boiled at a central school-meals kitchen in a copper vessel. Immediate vomiting followed later by diarrhoea which may last for hours are the usual and harmless symptoms, though one man had severe diarrhoea for three days. He, his dog, his wife and three children were all poisoned because they ate a loaf of bread part of which was green. They thought the green was merely mould when it was, in fact, caused by particles of bronze which had got into the bread from the baking machinery (51). A less acute

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is, however, no convincing evidence that aluminium in the amounts in which it is likely to be consumed as a result of using aluminium utensils has a harmful effect upon the ordinary consumer. It is possible that there may be individuals who are susceptible to even such small doses of aluminium as may be derived from aluminium utensils, but evidence of this is inconclusive.'

Arsenical poisoning reached its apogee in England in 1900 when some 6,000 people in the north of England and the Midlands were severely poisoned, and more than 70 died, from drinking beer. This beer was contaminated with arsenic because it was made from sugar which had been refined with sulphuric acid derived from pyrites that contained arsenic. The pandemic of poisoning continued for many months undiagnosed, sufferers being considered to have alcoholic neuritis or some form of dermatitis, including herpes zoster. Indeed the correct diagnosis was not achieved until Dr. Reynolds (57), pondering on this apparent epidemic of herpes zoster and of alcoholic neuritis, decided that as arsenic is the only known cause of herpes zoster—apart, of course, from the sporadic 'naturally' occurring infection—and as it also causes neuritis, his patients should be examined to see if they were excreting arsenic. From this brilliant application of an erudite piece of medical lore the explanation of the outbreak was revealed. Once revealed everyone recognized at first glance the arsenical puffy face, the hoarse voice and the limp. Since then the stringent enforcement of the regulations controlling the amount of arsenic permitted in food has virtually abolished arsenical food poisoning, apart from agricultural arsenical pesticides, etc., being left on apples or other foods (p. 96). But even so arsenic still crops up in the oddest circumstances, such as occurred recently in Rome (58). There the U.S.A. ambassadress ascribed various symptoms, including a weak foot

when dancing, to arsenical paint on her bedroom ceiling falling into her morning coffee. She had noticed that her coffee always tasted bitter even after she had substituted U.S.A. coffee for Italian.

Oleander (*Nerium oleander*) is a most poisonous shrub, but even so it is startling that in Australia 300 soldiers were ill after eating meat cooked on skewers of oleander and many died, while in another tragedy 7 out of 12 died—even stirring porridge with oleander twigs has caused poisoning (59). This, in England, would be only of academic interest were it not for the chance that campers might try emulating the cookery of the tough Australian.

Wrappers round food are a potential source of poisoning even when they are not deliberately impregnated with fungicides, etc. (p. 100). Thus the British Plastics Federation (60) has enumerated some 120 substances which might be used in making plastic wrappers or containers for food, and has pointed out that 30 of these are known to be toxic, including trinonyl phosphate, trixylyl phosphate, cadmium and lead and strontium naphthenate, barium and lithium and magnesium stearate, dibutyl tin dilaurate, etc. A further 15 are 'materials concerning which partial restrictions have been suggested regarding their use or regarding which their appears to be some doubt concerning the advisability of their use'. Any restraint in the materials used to make plastics is, with few exceptions, purely voluntary and 'the injudicious choice of other raw materials for plastics intended for contact with foodstuffs may lead to accidents which may cause damaging prejudices against the industry as a whole'. This is a sincere but possibly tactless reason for not poisoning the public. The problem is complicated by a plastic pipe, for instance, being suitable in a factory making bland pea soup but being unsuitable when the factory starts making an acid synthetic lemon cake-filling.

classified in 1954 were by 1955 reclassified as 'Colours which have been shown, or are suspected, to have harmful effects on health' (202). This tendency to become more dubious about the safety of dyes as more evidence is acquired about their cancer-causing properties is seen in other countries: in the U.S.A. (62) three of the 18 hitherto 'permitted' dyes were banned in 1955 and both Germany and France have also contracted their 'permitted' lists. Further, there is no universal agreement about what dyes are safe. Thus naphthol yellow S, considered in England to be one of the safer dyes, is considered by the International Union Against Cancer to be 'unsuitable or potentially dangerous which should not be added to food or drink for man or animals' (203). Nor do even two countries so akin scientifically as England and the U.S.A. have the same 'permitted' dyes: the U.S.A. has 15 dyes, England 30; yet we ban 9 of the U.S.A. dyes and in return have most of ours banned. This complete disagreement about the safety of individual artificial colours must mean that there is so little known about them that there is no satisfactory proof about the safety of any (163).

Why so little is known about the cancer-causing properties of dyes and, indeed, of other chemicals added to food is because chemicals which cause cancer act in the body in an entirely different way to those 'ordinary' poisons which have been considered up till now: as is fully discussed in the chapter on cancer (p. 117). They have no safe 'threshold' dose—no dose below which it is safe to consume them over long periods—and further there may be a 'latent' period of many years between their last consumption and the appearance of cancer. Hence when cancer does appear in man it is almost impossible to trace if it has been caused by a particular dye or other chemical in food: a man dying of cancer in 1969 when he is fifty years old will certainly not remember, for instance, what brand of margarine his wife

gave him to eat in 1949 when he was thirty, nor, even if he remembers, will anyone know if the brand then contained —as it might have (204)—a cancer-causing dye. Even were virtually the whole populace given a new cancer-causing dye in ices and even were there scores of thousands of deaths from cancer 15 to 20 years later, yet the lethality of the dyes in the ices might never be realized: the more people eat a noxive food the fewer are left to show, by contrast, its danger. As it is at present, therefore, proof from man that certain chemicals are cancer-causing is only available for the few which cause cancer in the men who manufacture them.

So an opinion as to whether a colour, etc., will cause cancer has to be based partly on whether its chemical composition is akin to that of chemicals already known to cause cancer and partly on animal experiments.

The chemical composition of dyes and other substances which cause cancer is a complicated subject, meaning little to anyone who is not an organic chemist. So the structural formulae of the various chemical classes involved and a list of the dyes permitted in foods in England is given in the Appendix (p. 152). The interested reader should consult the very lucid reviews given by the Food Standards Committee (201), by Haddow and Kon (205) and by Bonser and his colleagues (206). The first of these, only costing one shilling, can be bought from the Stationery Office, York House, Kingsway, London, W.C.2: it also covers the whole problem of dyes in foods. It is sad that what is a unique guide to the domestic prevention of cancer is unknown to the general public.

The essential chemical problem when considering whether substances can produce cancer is what change they undergo in the body. Probably few if any substances added to food are themselves capable of causing cancer: what the body turns them into is the danger. But very little is known about

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Colours and Investigation of Cancer-

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impossible to show it is safe (163).

It is not wise to eat any artifically colou
jams, iced cakes, sweets, ices, canned
custard powder, etc.

even when they carry slight risks, but, conversely, other methods do need to be altered or even given up, this being especially so of some of the more recent. It is essential that more attention should be paid, than has been usual in the past, to the possibly harmful effects of new procedures before they become so widely established that they form an integral part of the provision of food for concentrated populations.

Preservatives and Antioxidants

Food goes bad because of the growth in it of bacteria and moulds or because the fat in it becomes rancid through oxidation.

Preservatives in foods and drinks, in the sense of chemicals which destroy or inhibit the growth of bacteria and moulds are meticulously controlled by legislation. Most foods are not allowed to contain any preservatives beyond those traditionally used such as common salt, sugar, spices, etc.: bacon and ham may contain nitrates and nitrites and the substances added during smoking, the latter also being allowed in smoked fish: benzoic acid or its salts are permitted in small amounts in fruit pulp, fruit juice, non-alcoholic wines, mineral waters, coffee extracts, pickles, etc.: sulphur dioxide or sulphites in fruit pulp, jam, cider, beer, and dried fruit and vegetables; diphenyl or ortho-phenylphenol (p. 151) are allowed within prescribed limits when applied to the skin of oranges and other imported fruits to prevent the growth of moulds. None of these preservatives is directly harmful to the body used in the prescribed quantities, however indirectly objectionable they may be in, for instance, enabling jam manufacturers to keep fruit pulp for months before it is used for jam-making, its loss of colour being compensated for by the addition of artificial colour (p. 43).

Illegal use of preservatives is probably very rare, though oranges and their wrapping paper are sometimes still found to have been treated, to protect against moulds, with thiourea (p. 151) which in animals is not only toxic for the thyroid but also causes tumours (63).

Sorbic acid has recently been found to be an extremely efficient substance for preventing moulds growing in foods (64) It can be mixed with the food, sprayed on to the outside or put on the wrappers of food. It is very safe and has a simple chemical formula (p. 149) which does not suggest any possibility of toxicity. It seems probable that it will be permitted in foods and their wrappers.

Boric acid or borax, formaldehyde and nitrites are all illegal in fresh meat. but it seems they may all be used surreptitiously by butchers to prevent putrefaction. One patient of mine, a butcher's assistant, was given a powder to preserve meat though he did not know its composition; I have been told formaldehyde is used; nitrites cause a thin layer of pink on the outside of meat after roasting. This pink must not be confused with the pink layer which sometimes, especially in veal and mutton, may spontaneously occur during cooking: the particular shade of pink induced by nitrites will be recognized by housewives if they once soak a morsel of meat in a solution of sodium nitrite, bought at a chemist, and then roast the meat. All of these substances, in the amounts liable to be eaten with meat, are very unlikely to do more than cause an obscure diarrhoea, especially in children.

Penicillin and, better, some of the other antibiotics can be used for controlling the growth of bacteria in food. There is considerable commercial pressure that this folly should be permitted. The subject is discussed on p. 77.

Antioxidants are essentially used to prevent fats becoming rancid. All fats and especially those containing the essential

unsaturated fatty acids—'E.F.A.' (p. 69)—tend to become rancid by reacting with the oxygen in the air. The substances formed not only give food a horrid taste but are toxic, at least to animals (65). At present antioxidants to prevent this rancidity are not allowed to be added to foods, but since 1954 The Foods Standards Committee (66) have urged the permitted use, in prescribed amounts, of propyl or octyl or dodecyl gallate (p. 151), butylated hydroxyanisole and also (67) butylated hydroxytoluene (p. 151).

The first three appear to be safe (63, 66) but I have found *no research dealing with the safety of the last two*, apart from a little on butylated hydroxytoluene which needs confirmation (76).

Cooking, Canning and Freezing

Fats exposed to unusually high temperatures, such as may *sometimes occur in domestic cooking, develop cancer-*producing properties. This possibly might be of importance since 'the high incidence of gastric cancer in human beings as contrasted with the very low incidence in all other species . . . points essentially to some human habit or attribute as of aetiological importance. The variation in incidence . . . points rather to habits than to inborn human qualities as being mainly responsible . . . *methods of cooking* that involves heating fats and oils to temperatures of about 300° C. provide a source of potential carcinogens' (68). The temperatures of fats used for deep fat frying probably do not exceed 250° C. or 480° F., but frying in an open pan and roasting and grilling may expose fat to higher temperatures, especially if some of the newer frying fats are used which give the best results at temperatures higher than those needed with natural fats.

Gastro-papillomatosis, chronic gastric ulcers and fatty

degeneration of the liver, and cirrhosis, have also been caused in animals by heated fats, which also develop an anti-vitamin A factor that destroys part of the vitamin A activity of foods eaten at the same time (68). This factor would tend to increase in fats heated many times: a strong argument against deep fat frying where the fat is used again and again.

But since there are so many possibly injurious substances in food which can be avoided by anyone with far less trouble than is involved in altering succulent traditional cooking habits, it would seem far wiser and easier to be content to spend all the energy one can afford on refusing coloured foods, margarine, 'cooking fats', etc., than on refusing fried or roasted foods.

Greasing of baking tins for bread with natural fats or polymerised vegetable oils (69) has also been held to cause changes in fats which might be cancer-producing and the same applies to the use of liquid paraffin (62): hypochondriacs and academic purists should use baking tins and frying pans on which a thin layer of silicone has been fixed which prevents food sticking to the tins (70). Such films are very durable and appear utterly safe, as are polytetrafluoroethylene films (261).

Commercial canning and *domestic pressure cookers* do not involve temperatures which are dangerous from the present point of view, though the urine may smell unpleasant after eating canned meat, due apparently to aromatic substances being formed during the canning.

Pasteurization has been accused of many sins but no one has yet suggested that it causes the production of injurious substances.

Freezing, chilling and *cold storage* of meat and other foods can reduce their nutritive value but do not produce toxic substances. In passing it is interesting to note that the pre-

cooked frozen foods which are now widely sold in germ proof hygienic plastic bags must be eaten *immediately* whether they are meat, fish, pudding or vegetable. This is because the pre-cooking will not have killed the heat resistant *Clostridium botulinum*. This bacillus will lie dormant as long as the food remains frozen but, once thawing takes place, will multiply rapidly because all the other bacteria which normally prevent it growing, have been killed in the cooking and have been prevented from re-infecting the food, as happens in ordinary kitchens, because of its germ-proof wrapper (71). The toxin of this bacillus is deadly: it caused, for instance, the fatal tragedies at Loch Maree: those who had eaten the infected duck paste were at first gigglingly amused that they saw double without having had the expense of buying whisky. They died.

The lack-lustre eyes of fish show they are stale, but the white opacities that develop in the eyes of fish which have been frozen and then thawed are only recognizable by an expert (72) and so are of little help to the housewife trying to tell if fish are fresh.

Irradiation

Various forms of irradiation have been used or suggested for the preservation or cooking of food. Of these, infra-red and ultra-violet rays have a limited effectiveness and do not penetrate deeply enough into food, etc., to give them more than a limited commercial value. The changes they produce in food are probably innocuous, though the cancer-producing effect of ultra-violet rays on living animals could conceivably cause exposed food to become carcinogenic (62, 73). Ultra-sonic waves can be used for killing bacteria, etc.: they act by inducing extreme fluctuations in mechanical pressure and have a very limited and harmless use.

Ionizing radiations, however, are thought to be potentially of great value in sterilizing any food fresh or canned; in stopping stored potatoes sprouting; in stopping fruit ripening. Their destructive effect on many of the essential ingredients of food is extensive, complicated and far from fully understood. This destruction is accompanied by the formation of substances which again are not understood and may be toxic. Hannan (73) has written an extremely interesting and documented review of the whole subject which cannot be compressed: it must be read in full. But three quotations can serve to sum up how dangerous the ionizing radiation of foods may be and how very unwise it would be to tolerate its commercial use on the vast quantities of different foods which would be necessary if irradiation were to be economically feasible. The need for investigating on animals firstly the dietetic deficiencies caused; secondly the acute and chronic toxic chemicals probably formed; thirdly the *carcinogenic* or *cancer-causing* substances possibly produced—all these investigations properly done would bankrupt the laboratory facilities of the country (p. 46).

'Great difficulty must be expected in showing that any chemical changes occurring during or after irradiation do not result in the production of toxic or other harmful compounds'

'... the absence of carcinogenic effects in particular will need to be demonstrated and in this connection at least two types of chemical change can be noted:

1. Branching could be introduced into fatty acid carbon chains....

2. The possibility of changes in the steroids introduces potentialities for biological activity which are, as yet, only a field for speculation....'

'... the difficulties are enhanced by the known fact that ionizing radiations are potentially carcinogenic when

applied to living animals, even at very low dose levels. There is no positive indication that this effect is produced through the medium of a stable carcinogen or that such a carcinogen, if formed, would be transmissible to a consumer of the irradiated tissues, but the possibility cannot be discounted until exhaustive testing has been carried out.'

The U.S.A. Army had planned an enormous plant for irradiating food within plastic envelopes, thus enabling it to be kept virtually for ever. But the plant has been cancelled, owing to the injurious effects of irradiated food on animals (253).

Emulsifying and Stabilizing Agents

These agents are widely used in making bread, confectionery and other foods. Essentially the emulsifiers break up fats and oils into a cream which can then be mixed into cakes, etc., while stabilizers prevent such creams from reverting to fat or oil. Domestic examples of emulsions are mayonnaise, where olive oil is emulsified with the help of egg yolk; or ordinary cream which is a butter-fat emulsion caused and stabilized by the other ingredients in milk—churning cream to make butter breaks down the emulsion. Domestic emulsification is generally caused by eggs or by 'creaming' butter. In the commercial preparation of food, other and cheaper substances are mostly used. What these are is generally known, though at present there is no law which prevents a manufacturer using any substance he likes and keeping its composition and its presence in his foods a secret. The Foods Standards Committee (69), in the face of bitter commercial opposition, has urged that there shall be a 'permitted list' of emulsifying agents, etc., which may be used in food and not, as at present, a 'prohibited list'. Such a list merely bans substances after they have been proved

toxic, which means much harm could be done before the ban was effected.

The substances thought to be safe by the committee are all those of natural origin which are natural constituents of certain foods or are common food ingredients apart from their emulsifying properties. As well as these are another eight which are very like natural fats, etc., and are believed to be harmless. Then there are six considered to be safe though complete proof is lacking: these are considered satisfactory additions to food. In addition nine substances or groups of similar substances at present known to be used are considered unsatisfactory on the available evidence. Of course the committee could not report on any chemicals used covertly and not reported. This is worrying.

Of the six chemicals believed to be safe only one, a silicone used to prevent foaming in some food manufacturing processes—such as the production of evaporated milk, concentrated fruit juice or caramel colour—would seem to be dangerous. This substance, in itself, is physiologically inert and not absorbed from the intestine. But in one experiment on animals where silicone was fed with an emulsifier—itself harmless—the silicone severely damaged the liver, the kidney and the middle ear. The amount of silicone given was only 25 times as much as that which may be added to foods: yet the general belief is that substances fed to animals are only safe for man if they produce no injury in amounts 100 times greater than that added to human food. The microscopic appearances of the organs of the animals suggested that the silicone had been deposited within the cells (74). The animals were all killed after a few weeks, so no one knows what further damage the silicone might have caused after a longer period. But it is known that a silicone film, when implanted directly into the tissues, can cause cancer after a long latent period (75). This strongly suggests that silicone absorbed

from the gut and then deposited in the cells would, after a long time, cause cancer. Such absorption of silicone is now bound to occur, if any is present in the food, because it will meet the modern highly efficient emulsifiers added to so many foods. It is clear that silicone should not be used in the manufacture of foods, especially as those in which it is mostly used are those consumed in large amounts by young children.

The nine emulsifying substances, or classes of chemicals, not considered safe in food but now used, or possibly used, in the making of bread, confectionery, imitation cream, Kosher foods, margarine, flavouring compounds, etc., include lauryl sulphate and sodium dioctylsulphosuccinate (264) and a variety of polymers. Polymers are molecules of high molecular weight containing units which repeat themselves—the silicones, the polyoxyethylene derivatives, polyglycols, polyglycerols, polymerised oils and polymerised fatty acids. Many polymers are suspect from the point of view of causing cancer (69, 75) and some of the widely used polyoxyethylene derivatives are also toxic, at least to animals. 'The harmful effects observed in animals fed polyoxyethylene-derived surface-active agents at dietary levels as low as 5 per cent indicate that considerable caution should be exercised before such substances are used in foods for human consumption' (77). The effects induced in rats or hamsters (77) include decrease in growth and in final adult size, poor food utilization, unthriftiness, increased mortality, persistent moderate or severe diarrhoea and bleeding from the genito-urinary tract. The kidneys, lower bowel, bladder and testes are damaged, and stones are formed in the bladder.

But more important than any of the above effects of polyoxyethylene derivatives is the way they alter absorption from the intestine. They cause abnormally large amounts of

iron to be absorbed from ordinary foods, at least in animals, leading to its being deposited in the caecum, spleen, and the liver where it may cause cirrhosis (77) and cancer (98). In man it is known that both the rapidity of absorption of vitamin A and also the amount absorbed may be vastly increased, so that fantastically high levels of vitamin A are reached in the blood (83). It cannot be considered satisfactory thus to overwhelm the storage capacity of the body and, conceivably, overwhelm the placenta so that it lets through to the foetus abnormal amounts of the vitamin. Other normal substances besides iron and vitamin A must also tend to be absorbed too quickly and possibly in toxic amounts. Added to all this is the danger, known to occur in animals (74, 84, 264), that cancer-causing substances normally not absorbed are absorbed.

Artificial emulsifiers should not be used in foods.

Sweeteners, Flavours, Tenderizers

Sorbitol (p. 150) is a sweetish substance, chemically akin to glucose, found in scant amounts in fruit. Probably it is poorly absorbed (79) and what is absorbed is possibly largely excreted unchanged in the urine (78), so that its use as a sweetening agent for diabetics rests on its being nutritionally inert rather than on its being a safe nutritive substitute for sugar (79). Recently it has become so cheap that it is used in confectionery for a sweetener and plastifier, and to conceal the staleness of cakes by acting as a humectant to keep them moist. But one ounce tends to cause wind and, in about 6 per cent of people, discomfort and diarrhoea: so people suffering from wind and diarrhoea should remember this may merely be the result of eating shop cakes (80). Another humectant now, presumably, no longer used is diethylene glycol which in large amounts is acutely toxic in

man to the liver and kidney and, in animals in small amounts, causes tumours of the bladder.

Saccharine is the only other sweetener in use: it is harmless for rats and, to judge from its incessant eating by diabetics, for man. All other synthetic sweeteners are banned by law, including those which never were widely used in England—Dulcin, P4000 and Sulphamate or Cyclamate: all cause general toxicity in animals, with the first also producing liver tumours, the second damage to the kidney and the last diarrhoea (82).

Flavours—the formulae of some being given on p. 155—are generally a mixture of the natural and the imitation, since the natural are often not strong enough to be used alone in sweets, cakes, cake-mixes, ices, jams, soft drinks, etc. In the U.S.A., and so possibly already in England, some 300 flavouring agents are known: whether they are all safe is unknown. Little has been written about flavours, presumably because they are valuable commercial secrets. Many are, apparently, compounded from often simple and, in small amounts, innocuous chemicals producing such flavours as blackcurrant, raspberry, cherry, walnut, grape, butterscotch, butter, etc., while others, such as the amyl acetate of pear drops, are really new flavours. However, some of the *known* substances which often are used in a variety of flavours may be toxic: thus *Citral*, which occurs naturally in lemon peel, has been held to be, in part, the cause of citrus fruit poisoning (p. 103); *Glycyrrhetinic Acid* (262), the corticosteroid-miming acid made from the *liquorice* so beloved by children in the little back streets, is perturbingly akin in its chemical structure to the cancer-causing hydrocarbons (205); *Coumarin*, the naturally occurring poison in sweet clover with a taste like bad *vanilla*, is a cumulative haemorrhagic, hepatic and renal poison (233, 260), which is excellent for destroying rats—in the U.S.A. it

is banned as a flavour for food, in England it is used; *Honey* flavour may be obtained by the reaction at elevated temperatures of a sugar like glucose with beta-phenylalanine (257)—I have found no evidence about the safety of this 'honey'.

Tenderizers of meat are a vague class: glutamates are essentially natural substances the value of which is chiefly to enhance the taste; vinegar may make steaks more tender but is chiefly used in restaurants to remove the taint of early putrefaction; papain is a harmless vegetable enzyme which, well forked into steaks before cooking, partially digests them and totally ruins them gastronomically.

bromine: both combine with methionine and other nutrients in the wheat to form substances which may or may not be toxic: virtually no research has been done on this (157, 158, 159). Other insecticides (p. 90) may also contaminate the wheat because they were used on the farm during its growth, or they may be directly added to the stored wheat or be used to impregnate the walls of silos or grain bags.

Flour is, at its simplest, merely wheat ground up into a powder to facilitate its cooking and conversion into bread or the more complicated floury foods such as pastry or cakes. But simply taking any wheat available and grinding it between two stone millstones produces a dark 'whole' meal flour, of uncertain bread-making quality, which contains everything desirable or undesirable, present in the original wheat: from the wheat has been 'extracted' everything, so that '100 per cent extraction' flour is the result. Most people do not like such flour because it makes a heavy dry bread, often indigestible with a laxative effect. Therefore lower extraction flours are preferred: in the simplest and best, less flour is extracted from the wheat because the brown indigestible cellulose, the bran covering the wheat grain or wheat berry, is removed from the flour and fed to pigs or added as a laxative to breakfast foods. Such bran-free flour is certainly a better food, in spite of General Smuts and the philosophy of 'wholeism', the philosophy of eating the whole of food—the core with the apple; the hooves, horns and hide of the steer with the sirloin.

But having gone as far as removing the bran from the flour, having achieved roughly an 82 per cent extraction flour of a light cream colour, millers have unfortunately gone further and have produced a 70 per cent extraction flour: the dead white uniform flour of today.

Such white uniformity has been reached by the spur of commercial avarice driving the mule of research along

tracks better unexplored. The uniformity of bread, the dullness of bread, is achieved by blending together different wheats, such as the 'strong' wheats of Canada and the 'weak' wheats of England, so that their resulting mixed flour will produce under standardized mechanical baking a standardized uniform loaf.

But even such depressing uniformity in the baking quality of flour could not be maintained were the flour, once milled, loosed haphazard on the bakers

For the age of the flour is vital. Flour improves in its bread-making qualities as it ages. Therefore it is necessary, to achieve safe mediocrity, that all flour should be the same age. But this involves storage, and storage is costly and inconvenient. Therefore flour is 'aged' or 'improved' by chemical 'improvers'—like the agene of contentious memory—instead of being stored and so 'aged' in scattered bakeries throughout the country Whether this avoidance of scattered stores of flour all over the country is wise may be doubted, in view of the risk of strikes or atomic bombs sealing off central stores.

The colour of flour is again infuriating if dead uniformity is desired. But an all-merciful Providence has arranged that the very chemicals which 'age' flour also bleach it. Though there must be considerable doubt about whether the public really cares two hoots in hell about the colour of its bread: it certainly does not care to the extent of paying an extra 2½d. a loaf for dead whiteness, as was shown after the war when the off-white cheaper subsidized bread was bought in preference to the dead white unsubsidized bread (154). But the millers always claim that only dead white bread will satisfy the public and may, indeed, by now have hypnotized themselves into believing this. One wonders whether the desirability of whiteness was invented by the millers who, finding their chemicals bleached flour, had to defend

millers and health. Untreated 82 per cent extraction stone-ground flour may go sour, or breed insects, when carelessly stored or stored for too long. The sourness and, largely, the capacity to support insect life, is due to the oil in flour. This oil occurs only in the germ of the wheat. Most of the germ is got rid of by the steel rollers which have supplanted the stone millstones. These rollers crush the wheat instead of grinding it. While the brittle starchy part of the wheat breaks up into powder, the soft germ is only squeezed into flakes. These can be easily removed by bolting or sieving. So far, so good. A flour has been produced which is much less prone to going bad, at the cost of losing its germ and oil and E.F.A., and a large proportion of its vitamin B and vitamin E, its minerals and its essential amino acids. But even this is not enough. Some of the oil of the germ has escaped into the flour and the flour is still, probably, capable of going sour and of nourishing insects. Therefore chlorine dioxide has to be called in to finish off the flour's murder.

Chlorine dioxide destroys all the remaining vitamin E in flour (282), destroys or forms a toxic product (160) or a perverted one (164) with the oil's E.F A. (p. 69) and destroys (161) or may form a toxic product with the methionine. Add to this graveyard of nutrients a lingering miasma of chlorine dioxide and the millers have achieved a flour as nearly non-nutritious as is possible and as covertly, as insidiously corrupting to the body as food well can be. In the later chapters on human diseases it will be seen that bread twists like a scarlet garrotting cord round them all.

Mothers must make their family's bread, etc., from untreated stone-ground flour from small mills.

Margarine and Cooking Fats: Hardened or Hydrogenated Fats

'The Hydrogenation plants of our modern food industry may turn out to have contributed to the causation of a major disease.'

(Leading Article, *Lancet*, 1956, 2, 557)

Before the last war (162) all sections of the community—industrial, agricultural and rural—ate weekly per head 3 ounces of margarine and 6 to 8 ounces of butter, though the total amount spent on food was only 6s. 8d. to 9s. per head and margarine was extensively advertised, costing but 6d. per pound while butter cost 1s. 4d. To me it has always seemed that this refusal of all classes, in spite of the cheapness and clever advertising of margarine, to eat more than 3 ounces a week, must mean that there is some property of this fat which makes it injurious when more than a trivial amount is eaten. The present swing away from margarine to butter—as the war-enforced dietetic habits of an old insensitive generation are replaced by the free instinctive choice of a younger and so more sensitive generation—appears to confirm or at least emphasises the importance of reviewing what may be the injurious substances in margarine which, in a society with a free choice of food, cause it to be avoided.

All margarine and cooking fats are not the same: ingredients and methods of preparation differ, so the drawbacks may equally differ. But in essence margarine and cooking fats, domestic or commercial, are made from vegetable oils sufficiently 'hardened' or hydrogenated to give a fat of the desired hardness. The vegetable oils are liquid oils and not solid fats because they contain a higher proportion of essential unsaturated fatty acids or E.F.A., that is, certain fatty acids the molecules of which contain

two or more particular carbon atoms not saturated with hydrogen. 'Hardening' oils, or adding hydrogen to their unsaturated fatty acids, is brought about by heating them with hydrogen at high temperatures in the presence of minute amounts of nickel or some other catalyst.

The dangers of margarine are:

(*a*) Colouring may not be with the natural and safe carotene, as it should be, but with one of the 'permitted' synthetic dyes (p. 43) which in theory do not, but just possibly may, cause cancer.

(*b*) The original oils might, most improbably, be contaminated with insecticides (p. 90).

(*c*) The heating of the oils might be high enough to produce cancer-causing substances (68, 163).

(*d*) The E.F.A. or essential unsaturated fatty acids are destroyed or changed into abnormal toxic fatty acids with an anti-E.F.A. effect (164).

E.F.A. are essential both for many of the physical structures of the body and for many of its biochemical processes, which is the reason why they were originally called vitamin F. For their proper functioning they need vitamin B_6 (pyridoxine) and vitamin E, both of which are destroyed in flour, though this should be our best source. A simple deficiency of E.F.A., such as could be brought about by a diet centred —as many are—round flour and margarine, is thought to be at least a contributory cause to neurological diseases, cancer, heart diseases and arteriosclerosis—all of which are discussed in the last chapters of this book—and also skin diseases (83), and various degenerative conditions (165) such as *arcus senilis*, cataract, caries, senile osteoporosis and arthritis.

But the abnormal fatty acids produced by 'hardening' are the real worry. The atoms in the molecule of an essential fatty acid are arranged in space in a particular manner

controlling infection, or even dangerous to sensitized patients.

Yet while doctors are being thus urged to use fewer antibiotics, farmers are being urged to use more not only for serious illnesses in valuable animals like cows, but also as a routine addition to the food of cheap animals like pigs and hens, merely to increase slightly their rate of growth or to permit their being kept in overcrowded, infectious, unhealthy conditions.

This universal agricultural use of antibiotics has led not only to resistant strains of bacteria being increasingly common in food but also to food itself now containing antibiotics. Though this book is only concerned with toxic substances, like antiobiotics, in food, the production of resistant bacteria is also discussed later since it is not reasonable to discuss only one half of a problem.

Milk is the outstanding example of a food contaminated with antibiotics. Cows frequently suffer from mastitis, an infection of the udder generally due either to a staphylococcal or to a streptococcal infection. This is serious as it causes a loss of milk. The treatment is the insertion into the milk ducts of the udder of penicillin or some other antibiotic. After the mastitis has been cured, the milk should not be sold for human consumption for at least three days, in order that all the antibiotic within the udder shall have been

heavily contaminated with antibiotic. For instance in Somerset (85) penicillin is found about once a week in bulked milk from several herds, the amount in milk from a single herd being as high as 28,000 units per pint.

Allergic reactions from drinking such penicillin-contaminated milk have been reported (86, 265); one man, for instance, developed a generalized rash with vesicles, en-

larged glands, swollen knees and ankles and a temperature of 101° F. Such reaction may not occur until fifteen days after drinking the milk, and since the milk on some days may be free of penicillin, it is probable that many obscure cases of recurrent dermatitis without any obvious cause are due to penicillin in milk. Though more serious reactions, such as asthma or shock, have not been reported, they certainly could occur since even sucking a penicillin lozenge has caused collapse. The U.S.A. has considered banning antibiotics for the treatment of mastitis (87).

A further drawback of antibiotics in food is that they may cause a patient not already sensitive to become sensitive to them, with the result that they cannot be given for urgent conditions, nor can any medicinal substance be used which contains even traces of antibiotics. Thus poliomyelitis vaccine, which contains penicillin and streptomycin, may relatively commonly cause a violent reaction (88, 265): one boy, known to be sensitive to penicillin, a few hours after an injection of the vaccine developed an itchy rash quickly followed by vesicles and by severe headache and general lethargy. Three days later he developed broncho-spasm and remained ill for ten days. Human milk will contain any antibiotics given orally to the mother (266).

Blue cheeses, like Roquefort, may cause penicillin reactions (265), not because the milk from which they are made contains penicillin but because the blue moulds of the cheeses themselves form penicillin.

Penicillin and other antibiotics have also been accused, on very thin evidence, of causing many other illnesses. 'It is doubtful whether any agent for iatrogenic disease has been as busy as penicillin' (92), this antibiotic just possibly being related to leukaemia (92, 93) and, together with other antibiotics, it has been held to increase both the risk and virulence of virus infections (89).

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antibiotics, and resistant strains have been found in the faeces of 36 per cent of pigs going to a bacon factory (95). Other bacteria, such as *Clostridium welchii*, also, of course, become resistant. In fact on farms, as in medicine, the antibiotics are, through their wanton use, losing their value so that for instance 'the incidence of mastitis is, on the whole, very little better than it was before antibiotics were introduced—and cases are much more difficult to treat' (96). Urban England could cynically ignore this farming problem were not our farms still the basis of our food and therefore unavoidably a possible source of human infection. Often it is impossible to trace the origin of a human infection to another person, yet the role of animals is commonly overlooked though cases of staphylococcal food poisoning have been directly traced to an infected cow's milk (97), and epidemics of salmonella poisoning from duck eggs are common. Fatal infections of the bowel with antibiotic-resistant staphylococci are now a major problem in hospitals and though these bacteria are mostly considered to have originated in the hospital itself, there is no reason why they should not have come, in the first place, from milk, especially as milk is the staple food of the ill. Again, in very young children diarrhoea from *Escherichia coli* is common and should respond to antibiotics, but already resistant strains are common in farms (95) and so could easily be the source of diarrhoea which fails to respond to antibiotic therapy.

Brucellosis or 'Malta fever' is now rare in England but it easily could become a serious problem both for man and animals (100), so that the risk of breeding resistant strains by feeding cows and pigs with antibiotics is too lightly ignored. Again it is worrying that in Australia (99) a new strain of resistant staphylococci has spread in the general community, causing septicaemia with over a 40 per cent mortality. Generally such resistant strains are held to be

bred in hospitals and to be largely limited to people in contact with hospitals, but when, as in Australia, the general community is involved it would seem most probable that the infection has spread from antibiotic-treated animals to farm workers to everyone. Or it may be that the spread is through dairy foods like milk or, unexpectedly, cheese, since in Canada, 'Our results suggest the possibility that where penicillin or other antibiotics are used with dairy cattle, the survival of resistant organisms may lead to widespread distribution of resistant strains into the homes of the general populace, since staphylococci and streptococci, often in large numbers, are of common occurrence in cheese. Foods so contaminated may well contribute to the severity of the problem arising from infections in man with resistant strains without the patients having received prior antibiotic therapy and without having been exposed to endemic infections in hospitals' (101).

Fungal infections in man may occur in the brain, lungs, gut, kidneys, skin, etc., and are now increasingly common since the antibiotics kill the bacteria which normally inhibit the growth of fungi. Most of these fungal infections have been fatal until now, or extremely unpleasant like the monilia infections of the vagina during pregnancy, following the treatment of pneumonia, etc., with aureomycin. Now there is some hope that the new antifungal antibiotics will cure such infections, but this hope is dimmed by the fact that farmers have forestalled doctors in the use of these antibiotics and presumably have already started to build up strains of resistant fungi, since resistance is rapidly developed.

Lastly, one must take a gloomy view of the chances of the Government resisting Commerce's demands to be allowed to add antibiotics to foods like meat, poultry and fish in order to check the growth of bacteria in such foods. The antibiotics will kill the bacteria which normally give a

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pies or stews, or make the foundation of soup, or are given to the dog. Commercially, surplus necks must often find their way back into costly human foods such as meat pastes, chicken jellies, etc. Used in animal foods they are, of course, equally noxious.

In cattle pellets, usually of 60 mg. of hexoestrol, may be implanted wherever is easiest, though farmers are urged to put them into the bullock's ear which later should be burnt in the slaughterhouse and not handed to the ever voracious food processors. Wether lambs, and old ewes being fattened for slaughter, are implanted with 15 to 60 mg. of hexoestrol wherever convenient, though in theory in the ear. Pigs are not improved by oestrogens which is fortunate since an implant anywhere would inevitably be eaten, pigs not being skinned and even the ears being, unforgivably, put by the parsimonious housewife—or the food manufacturer—into pigshead brawn (thus ruining the cheapest and most succulent of all domestic dishes)

The risk, the very definite risk especially with all poultry, of eating the whole of a pellet which may be almost as large as when it was implanted, must mean that many people may absorb very large doses of oestrogens· doses large enough to cause headaches, nausea, bloating, rashes, etc., in many people; doses 30 times as large as those given daily for short periods to menopausal women; doses of the same order as those given before a wedding to postpone menstruation or suppress lactation; doses causing bleeding, and so fear of and examination for cancer in post-menopausal women. Much smaller doses may decrease a man's libido and cause enlargement of his breasts—gynaecomastia—to a socially embarrassing extent, especially when cirrhosis of the liver has impaired his capacity to destroy oestrogens. Sexual development in young girls will be precocious, in boys perverted.

self which may do this rapidly or slowly, or may in the process of destruction form even more poisonous substances (109), as also may animals feeding on such plants (110). Of course there is no way, such as washing, which can remove a systemic pesticide from a plant.

To reduce the quantities of insecticides left in or on plants, warnings are given for each as to how long should elapse in between spraying, etc., and harvesting. Often such warnings must be ignored because of changes in the weather, the demands of the market and the other claims on the available labour. Also the amounts of pesticides sprayed on may grossly exceed what is advisable, so leaving a high residue. There is also the very serious problem of sprays from aeroplanes being blown on to neighbouring crops, animals, children and trespassers: the first of these may be rendered unfit for consumption but yet may be unwittingly harvested when, '. . . under conditions relatively common . . . it is perfectly possible for drops of a certain size to travel three to four miles and this is, I suggest, a matter of the utmost importance' (109).

Accumulations of chemicals in the soil may be evanescent or lasting: thus sandy soil may contain 74 per cent of the DDT applied eight years before, while DDT sprayed on orchards increased from 14 lb. to 61 lb. per acre in four years (109). Therefore plants grown in soils previously treated can be exposed to contamination for many years. Potatoes are not planted in a field for at least 18 months after BHC has been used on it because they may otherwise be so heavily contaminated that they have an unpleasant taste. Surely a better protection should be given against contaminated food than the consumer's sense of taste, especially when insecticides may be tasteless.

No control in England of the manufacture, sale or use of these chemicals is enforced, though there are regulations

governing the way in which farm workers must protect themselves with special clothing, gas masks, etc., when applying the more dangerous chemicals to crops. There are also no regulations controlling the amounts of such chemicals which may remain in or on the crops when sold.

This lack of regulations is due to there being for many chemicals no feasible analytical methods for demonstrating their presence in foods; further, some chemicals, having been absorbed by animals or plants, are changed into unknown and possibly more toxic substances. No regulations are worth promulgating if it is not possible to prove they have been broken. But the toxicity of all these chemicals for at least some forms of life is the sole reason for their use, so there is a *prima facie* case for considering them noxious to man. It would be wise to rule that no new chemicals may be employed unless:

(*a*) Chronic toxicity tests show them and their impurities to be harmless when fed to three species of animals, male and female, all their lives and to their offspring. rats and pigs and ducks would be suitable.

(*b*) Practicable analytical methods are evolved by the makers for measuring the amounts of such chemicals and their breakdown products remaining in foods.

(*c*) Their composition is known and published with the composition of all their chemical impurities: some toxic chemicals contain up to 88 per cent of impurities which may be even more toxic than they are themselves.

A disaster from not adhering to all the above precautions was in one case only averted by chance. An insecticide, 2-acetaminofluorene, with a completely new chemical formula (p. 157), was just about to be widely used when research workers on cancer, happening by coincidence to be investigating it, agreed it was indeed safe in the sense of not being *acutely* toxic to animals, but pointed out that it had

sucklings: adult rats and kittens are killed by the milk from goats eating DDT, and rats are made ill by the butter (268).

Human milk in the U.S.A. has been found to contain up to 0·77 p.p.m. of DDT and human abdominal fat up to 34 p.p.m. (113), all presumably from food which in the U.S.A. contains on the average about 0·2 mg. daily (114). On experimental diets containing about 200 times this amount men stored in their fat up to 400 p.p.m. after 18 months, or 3·28 gm. in their whole body (114). They developed no abnormal symptoms nor was their liver function impaired.

This excretion in milk and storage in fat is worrying: though, at least in dogs, the foetus appears to be protected from insecticides absorbed by its mother, yet when it is born it is killed by its mother's milk (115), being apparently either abnormally sensitive to or poisoned by a highly toxic derivative of these made and excreted by its mother (110). In man quite different results, of course, may occur to those which occur in animals: at worst the foetus may be damaged, at best the milk may not be toxic. But one cannot be happy about exposing the growing foetus and infant to such possible risk (p. 111).

In illness, especially when it is prolonged and serious, most of the fat of the body may be rapidly used up. This would mean that there would be a sudden liberation within the body of DDT which, from the above figures, might be as much in all as 4 mg. per kilo of body weight—a dose which is uncomfortably close to the dose of 2·5 mg. daily which is said to be toxic for healthy men (114) and which presumably would be far more toxic to a body already labouring under severe illness. Such a liberation of DDT and subsequent toxicity occurs in animals when they are starved after consuming DDT (116).

The highly chlorinated naphthalenes (117, 118) form an interesting tail to this group of chemicals, showing how varied

animal's body and are not excreted in milk: but this can be questioned because animals and man may not show symptoms of paralytic poisoning from them until perhaps 10 days after being poisoned (123, 124, 125), which suggests their persistence within the body and their slow transformation into fresh toxic substances.

Cholinesterase is destroyed in the human body by all these insecticides. Cholinesterase is the enzyme which, by converting acetylcholine to choline, prevents the toxic accumulation of acetylcholine at nerve endings and synapses, and so prevents the over-stimulation of muscles and glands by acetylcholine. The symptoms of acute poisoning appear to be adequately explained by this destruction of cholinesterase and consequent toxic excess of acetylcholine: contraction of the pupils, headaches, photophobia; bronchial spasm; abdominal pain, nausea, vomiting, diarrhoea; muscular weakness, twitchings, convulsions, asphyxia, death. If men or animals survive one or even several severe poisonings, they completely recover, with the exception that very occasionally recovery from coma is followed later by relapse and death for no understood reason, or some ten days later permanent paralysis, discussed below, occurs.

But even though acute poisoning thus appears to hinge on destruction of cholinesterase, other less obvious damage is done, which may be more important from the point of view of covert prolonged poisoning from food: for instance the enzymes chymotrypsin, trypsin, liver esterase, milk esterase and succinoxidase are destroyed; and, more serious, there is probably a profound disturbance of amino acid metabolism (109). I cannot overstress my belief that in the future unexpected insidious damage to many organs will be found to be due to protein metabolism, essential amino acid metabolism, being deranged not only by insecticides but also by other chemicals, like those used to treat flour, present in

fiable substances before they have insecticidal activity or toxicity: parathion's activity and toxicity is really due to one thousandth of it being converted into paraoxon (109); what toxic substances are formed from schradan, systox and malathion, etc., are unknown (109, 120, 121). This ignorance about what this group, commercially, may consist of, and about what toxic products are evolved by plants and animals, is perturbing and largely vitiates the value of measuring residues of the insecticides themselves left in foods: the absence of any insecticide does not prove the absence of highly toxic unidentified substances derived from it.

Plant foods are those most likely to be contaminated, due to plants being sprayed close to harvesting: thus in France in 1955 acute poisoning occurred from eating peaches, melons, tomatoes, beans, etc., sprayed with demeton (109), and this insecticide has been found in orange juice, apples, pears and nuts when these were sprayed 4 weeks before harvesting—amounts ranged from 0·007 to 0·102 mg. per kilo (109). There may be 0·5 mg. of parathion in a kilo of tomato juice (109), while Greek olive oil has been found to contain up to 14 mg. of parathion per kilo, and local inhabitants using this oil had low levels of cholinesterase in their blood; French wine may contain 4 mg. per kilo (109). Crude cotton seed oil may contain 88 mg. of schradan per kilo (109). Stored grain—and its flour—dusted with malathion (121), for instance, might contain large amounts of pesticide if only cursorily cleaned. Cocoa beans from trees treated with dimefox were found to contain 0·1 mg. per kilo; rats and guinea pigs refused to eat them; food plants grown in the same area appeared to contain significant amounts (122).

Meat and milk are said not to be contaminated with these insecticides because they are rapidly destroyed within the

than 6,000 Moroccans who had used cooking oil adulterated with surplus U.S.A. Air Force aviation oil (276).

Paralysis has also been caused in dogs (131) by repeated dosing with DFP—a near relation to the insecticides—while in rats and rabbits (124) weakness and demyelination has been caused by mipafox, DFP and TOCP. All these three have caused demyelination and paralysis in hens, which are also atypically paralysed by malathion and EPN (132).

All the above paralysis-causing chemicals share with the organo-phosphorus insecticides the property of destroying cholinesterase. This destruction is not, however, itself the direct cause of the paralysis (124). Yet the worry must remain that this destruction may be a pointer to a second parallel destruction of enzymes essential for the integrity of the physical structure of the myelin (p. 130) of the nervous system. With most insecticides this second destruction is so slight that before lasting damage is done the healthy body has remade the destroyed enzymes. But freedom from damage must depend on this rapid remaking: there are conditions where enzymes might not be remade rapidly enough to salve the nervous system, particularly where it is already liable to demyelination. Thus one may especially fear that the effects of these insecticides, eaten perpetually in small amounts in foods, will become overt in those who, by heredity, have little cholinesterase (133) and so may also lack other enzymes; in those who, because of cirrhosis of the liver, etc., have low stores of cholinesterase—and presumably of other enzymes—and cannot adequately replace that destroyed (134); in those who are prone for no known reason to the demyelinating diseases or who have already developed them (p. 130).

our staple foods. There is also the nightmare suggestion that this perversion of protein metabolism might lead to the creation of new viruses (126).

A more obvious worry, however, is the demyelination of the nervous system (p. 129) with permanent paralysis, which may be caused in man by insecticides like mipafox (p. 156). The history of mipafox is worth the telling, since it exemplifies the dangers inherent in introducing new chemicals. In February 1951 *The Times* (127), *Nature* (128), and *The Farmer's Weekly* (129) variously described mipafox or 'Isopestox' as a new systemic insecticide 'much less poisonous to man' than schradan and capable of safe use even by the layman. For rabbits and guinea pigs it had only one twenty-sixth of the toxicity of parathion. Its special value lay in the safety of its application to plants such as hops, lettuce, spinach or Brussels sprouts nearer to harvesting than is possible with more toxic chemicals. Plants destroyed half of what they absorbed in 6 days and all in 16. In March (130) advertisements to farmers stated, 'Above all, Isopestox (mipafox) is safe for even the amateur to use.' In August (123) a man and a young woman manufacturing mipafox both developed the classical picture of acute poisoning with this group of insecticides (p. 91) and both completely recovered; some ten days later both developed severe paralysis, especially of the legs, which after two years did not improve enough to allow the young woman to stand unaided or the man to do other than a sedentary job.

Such paralysis appears to be identical with the often permanent 'ginger paralysis' which in 1931 afflicted some 15,000 Americans who had drunk 'Jamaica Ginger' adulterated with tri-*ortho*-cresyl phosphate or TOCP; contaminated soya bean oil used for salads and cooking in Durban and on board ship has also caused such paralysis (125). An apparently similar type of paralysis in 1959 affected more

have been sprayed with arsenic and to stress again that sodium arsenic causes cancer in man though in no other animal (207), as happened to one five-years-old boy who died from multiple skin cancers due to playing in fields, etc., sprayed with arsenic (142).

Of irrelevant interest is that cattle like arsenic, breaking into sprayed fields to eat it—recently more than 20 cattle on two farms in Somerset died from grazing in sprayed potato fields, which led the Chairman of the Bath National Farmers' Union, and other farmers, to state that these arsenical sprays are too dangerous to be released to farmers and 'without taking extraordinary precautions, which would not enter the head of the ordinary user, it is practically impossible to guarantee that human beings and livestock do not enter the sprayed areas' (143). The farm workers using the sprays presumably die of cancer of the skin and lungs more often than do other workers (145). The farmer saves 30/- an acre of potatoes by having arsenic used instead of other harmless haulm destroyers (291).

The amount of arsenic in sprayed potatoes—English or imported—is unknown: a Ministry investigation (141) in 1959 is worthless since the potatoes analysed may or may not have been sprayed—even so in 450 samples the peel of 21 and the flesh of 6 had over the 1 mg per kilo which is the legally permitted upper limit for arsenic. Further, the amount of arsenic in potatoes will presumably vary with the amount sprayed on them, the weather, type of soil, etc. An easy and genuinely valuable piece of research for an agricultural student would be to measure the arsenic in potatoes grown in various soils and sprayed under various conditions of weather.

Lead arsenate combines the drawbacks of lead (p. 35) and arsenic (p. 40). It is used chiefly as an insecticidal dust or spray over apples and vegetables such as cabbage and

cases of pink disease in infants and young children, nephritis at any age and that very odd condition of erethism which so closely mimics parkinsonism or paralysis agitans (p. 133).

Used to dress seeds; to control eel worm, club root, scab, etc., in potatoes and fungus infections of apples, pears, tomatoes, etc.; to fumigate stored grain and to kill moss in lawns, mercury can be absorbed from a number of foods. Probably the amount left on stored grain is harmless (138); there appears to be no investigation of mercury in potatoes; a child of two and a half years nearly died from mercury poisoning through playing on a lawn treated with lawn sand containing mercury (139); sprayed apples may contain 0·1 mg. per kilo (275); tomatoes 0·5 mg. per kilo mainly in the pulp and not as one would expect on the skin, which 'is a potential health hazard to the consumer' (140), and certainly most dangerous when the juice is given to weanlings, or adults eat copiously of their own over-sprayed tomatoes or drink bottled commercial juice.

Arsenic in all its forms should be banned from use in agriculture and horticulture but, very shockingly, there seems to be no hope of this (280). Arsenic as a solution of sodium or potassium arsenite is, until the 'voluntary' ban in the autumn of 1959, widely used in sprays from the ground or from aeroplanes to destroy potato haulm. Weeds in paths, hard tennis courts, etc., and among daffodils or tulips can also be killed, with the possible risk of contaminating nearby fruit and vegetables in market and domestic gardens. But of course the chief risk is from aeroplane sprays drifting on to neighbouring crops or on to the blackberries in hedges which so delight the potato-gatherers' children and urban caravanners, or on to the equally delightful field mushrooms—the only kind with any flavour. The poisonous effects of arsenic have been discussed on p. 40, so here it is only important to point out that imported potatoes may

have been sprayed with arsenic and to stress again that sodium arsenic causes cancer in man though in no other animal (207), as happened to one five-years-old boy who died from multiple skin cancers due to playing in fields, etc., sprayed with arsenic (142).

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Nonyl alcohol fumigation is used to check the sprouting of ware potatoes which presumably are not rendered toxic.

The fungicides (p. 161) chiefly used in agriculture—apart from those containing mercury or arsenic (pp. 95, 96)—are thiram, the oral toxicity of which seems unknown; ziram which is acutely toxic because of its zinc, its chronic toxicity being unknown; ferbam, said to cause nephritis in man and slight harm to animals when fed for long periods of time; zineb causing goitre both in man and in animals; salicylanilide of unknown toxicity (152). The risk to the public from fungicides, of course, lies in their persistence on crops such as apples or pears while sprays may accidentally contaminate neighbouring crops.

Molluscocides

Metaldehyde, better known to the public as 'Meta' solid fuel, is the basis of most commercial poisons against slugs or snails, and is often used by gardeners, ground up with sawdust, to make a protective ring round fruiting strawberry plants. It should not be used. It is intensely toxic. A girl of 3½ years (119) ate some metaldehyde-contaminated strawberries from a farm where a commercial metaldehyde preparation was used against snails: she developed pain in her arms, nausea, intense retching and repeated vomiting; she became very cold with cramp-like flexion of her extremities; she lost consciousness and became completely flaccid; her blood pressure was too low to measure, her temperature was 105·6° F. She did recover without developing the usual nephritis and atrophy of the liver.

Frogs or toads should be encouraged in strawberry beds to eat the slugs: the former will arrive of their own accord if a small water garden or old kitchen sink filled with water is put in one corner of the bed, arranged with stones so that

the frogs can get in and out, and surrounded by tall grass or plants to give soothing shade. Dogs must be forbidden to tease them. Toads have to be bribed to stay with bread and milk.

Copper sulphate is used in ditches and wet fields against the small snails which carry the liver fluke of sheep—an infestation which recently killed on one Cornish farm 60 out of 80 breeding ewes. Though copper sulphate will not render any foods toxic under these conditions, it is of interest that snails are best controlled by a flock of Khaki Campbell or Indian Runner ducks, including with them two white Aylesbury drakes in order to show the farmer's wife their whereabouts if they fail to return in the evening from their snail-eating orgy to their egg-laying duty (153).

CHAPTER XI

The Prevention of Dental Decay; Soft Drinks; Fluoridation

Dental decay is the commonest preventable di All children, with rare exceptions, have so: their milk teeth decayed before they are finall) (176), and about one third of all milk teeth are al decayed before the child is six years old (177). By f years of age more than 90 per cent of children have decayed permanent teeth, the average number of de(teeth being more than seven (176). Thus adult life is with a septic mouth incapable of proper mastication.

This pandemic disease of the teeth is chiefly due to mc *food manufacture adding to carbohydrate foods th* jurious quality of stickiness.

Sugars, sweets, the slowly sucked peppermints belov adults, *and soft sticky white bread, biscuits, cakes, etc.,* round the teeth. There their sugars and starches are de(posed by bacteria with the production of acid. This *dissolves away the hard protective enamel on the outsi(* the teeth, thus exposing the soft dentine beneath to bact *invasion. This invasion is the only cause of dental deca* caries.

Soft drinks, being often highly acid, also dissolve a enamel, thus reinforcing the acid made by the bacteria dirty mouth. Soft drinks are mainly made from the jı

peel, etc., of oranges or lemons sweetened with sugar; or from citric, phosphoric, tartaric or lactic acids together with sugar and flavouring agents and sometimes carbon dioxide to give fizziness. Quinine or caffeine are often added.

Orange and other citrus drinks, whether they are home-made or bought, attack the enamel because their citric acid is particularly injurious (178). 'Ready-to-drink' commercial orange drinks can be less harmful to the enamel and far less nutritious to the consumer than home-made ones because they may, legally, only contain in the usual 6 oz. bottle one-eighth of a teaspoon of orange juice or if the entire orange, peel included, has been ground up or 'comminuted', two-fifths of an entire orange.

Excessive drinking of orange juice and especially of drinks made from 'comminuted' oranges may be harmful beyond their effect on the teeth: one report (179) mentions headaches, constant physical exhaustion, abdominal pain, prolonged common colds, joint pains and loosening and hypersensitiveness of the teeth. This is borne out by friends of mine who say that in some parts of the U.S.A., where orange juice is served instead of water, visitors after a few weeks of guzzling the juice develop all the symptoms of chronic influenza. A hairdresser tells me that young women 'slimming' on a dozen oranges a day get grossly scurfy heads. The terpenes in the juice appear to be the cause of the gastric distress in some adults (180).

But it is the oil from the peel, squeezed out by mechanical pressing or included in 'comminuted' commercial drinks, which so upsets children that synthetic vitamin C has to be substituted for this 'natural' orange juice (181), while the flavonoid substances may perhaps cause a stress reaction (182). Of course drinks made from 'comminuted' oranges may also contain the fungicides, etc. (p. 50) and also the artificial colours (p. 43) with which the skin may have been treated.

The cola type of drink, assuming all these drinks are very similar, contains orthophosphoric and citric acids, extracts of cola and vanilla, caffeine, sugar, caramel, salt, carbon dioxide and commonly used essential oils, seven-eighths of which are citrus oils and the remainder spice oils (183).

Some English workers (184) have found the cola drinks not nearly so destructive to the teeth of animals as fruit drinks, while others in South Africa and the U.S.A. have reported that, 'The Cola beverages were found to be among the most acid and to contain 10 per cent sucrose and 0·055 phosphoric acid with a pH of 2·6' (185) and that there is severe destruction of the molar enamel after drinking cola for 5 days (186). 'In the beginning one of the common cola beverages was used in this study, but the molar teeth of the experimental rats were so badly eroded by the end of six months that the rats could not eat normally, so this experiment was terminated' (188). This eroding effect has been widely confirmed for cola drinks, or drinks with a similar phosphoric acid content, using rats, puppies, dogs and monkeys (175, 185, 187, 188). The injurious, horrid results of cola drinking on the enamel of the teeth have also been observed in man (189).

Other effects of cola drinks are obscure. Their popularity, and addiction thereto by small children, adolescents and adults, is difficult to explain merely by their reported analysis (183) which is generally not divulged or confirmed by the makers themselves. The quarter or half grain of caffeine in the usual 6 or 7 oz. bottle can hardly have an appreciable stimulating effect, though people who do not give tea to small children might be equally dubious about cola drinks if they realized their composition. Others again might feel that all the ingredients of so widely consumed a drink should be divulged to the Ministry of Health, even if not to the public whose concern they really, of course, are, since the

citrus oils, discussed under orange drinks, may be harmful; 'spice oils' is a vague description; extracts of cola are stimulating; acids rot the teeth.

Frozen-hard soft drinks, like *iced lollies*, and *acid sweets* and even some *coughdrops* (184, 189) all erode the teeth, and since they are sucked slowly their acids and sugars have ample time to act.

Light beer with a pH of about 4—as has cider—is not acid enough to damage the teeth and, having none of the other drawbacks of soft drinks, but containing instead many essential nutrients, is far healthier than soft drinks at all ages. It is sad that a crippling tax and foolish licensing laws have enabled untaxed soft drinks to compete with light beers.

The *prevention* of dental decay is simple: the mouth should be kept clean. No decay occurs unless there are both carbohydrate and bacteria in the mouth (191). Therefore no food, snacks, biscuits, sweets, etc , and no soft drinks should be taken between meals. After every meal the mouth should be thoroughly rinsed with water. children can be easily taught to do this surreptitiously and swallow the water afterwards. When being taught they should spit out the rinsing water into a glass so that they can see how much food has been dirtying their teeth and how much more they can dislodge from between the teeth by squeezing the water about with their cheeks and tongue

At night nothing should be eaten or drunk after cleaning the teeth and rinsing the mouth, while in the morning the teeth should be cleaned and rinsed *after* breakfast.

Infants should be breast fed: if bottle fed they must never be allowed to sleep with the teat in their mouth nor must dummies ever be allowed: '. . . the immersion of a rubber dummy in rose-hip or blackcurrant syrup before use with frequent replenishments throughout the day and night.

This, if continued for any length of time, results in a startling destruction of nearly all the teeth' (190).

Foods in general should be unrefined and hard: refined foods like white bread and biscuits stick round the teeth far more than wholemeal or home-made bread or real porridge; whole oranges are less erosive to the enamel than their juice (178); hard foods like apples clean the mouth by making children chew and by stimulating a flow of cleansing saliva.

If the public refuse to train their children and themselves to keep their mouths clean, then the only alternative is to try to increase the resistance of the enamel to dirty mouths and acid drinks. This can be done by adding fluorides to the drinking water. Whether this can check the increasing decay caused by increasing consumption of sweets, acid drinks, etc., is doubtful, even if such fluoridation is wise.

Fluoridation

Fluorides (32–42) are of considerable interest because their protective effect against dental decay has led to the current proposal to compel their addition to drinking water, bringing the total content up to about one part per million (1 p.p.m.).

Most untreated drinking waters contain about one-tenth of a milligram of fluoride per litre or 0·1 parts per million (0·1 p.p m.) while in some areas, such as Maldon in Essex, there may be 5 p.p.m. Fluorides cannot be removed by ordinary domestic water-softeners, though one difficult to use might be made.

The daily intake of fluoride when drinking 3 pints daily of water containing 1 p.p.m. is 1·7 mg. To this must be added the fluorides in other foods and drinks, though the reported amounts vary greatly and are at best a rough estimate: sea

fish, which eaten in large quantities (35) may themselves cause mottled teeth (*vide infra*), contain 5 to 10 p.p.m. and tinned pilchards 5·8 p.p.m.; flour, compulsorily fortified with chalk which is rich in fluoride, 0·7 to 1·0 p.p.m., giving a child of 12 years an average daily intake of 0·26 mg.; soup made from bones 3·6 p.p.m. or 1 mg. in an average helping; baking powders, etc., may provide 0 8 mg. daily. Other common foods, in the quantities eaten, do not appear to be important sources of fluoride, but strong beers may contain 2·3 p.p.m. and figures for dry tea of the cheaper brands have been given as high as 258 p p.m., which as drunk could provide 3 to 6 mg. daily. Most of the fluoride ingested is rapidly excreted, or stored in the bones and slowly excreted.

Dental decay is reduced by one half to two-thirds in children who have been brought up on water containing 1 p.p.m. or more of fluoride. This protection probably persists into adult life The fluoride has to be taken while the enamel of the teeth is being formed: that is during foetal life and early childhood. Fortunately women, unlike cows and some other mammals, pass on fluorides both to the foetus and the suckling when they themselves are taking fluorides. Accompanying this dental protection is mottling of the teeth of some children, though children who show no mottling are also protected. With water containing 1·0 p.p.m. of fluoride only 10 per cent of children show mottling and with 2·0 p p.m about 50 per cent. At these levels of fluoride the mottling is aesthetically harmless, taking the form of unnoticeable small white lustreless spots in the enamel. But above about 3 p.p.m. the white spots tend to coalesce and form blurred horizontal lines or large splotches which are desolatingly disfiguring on the front teeth, especially as they turn yellow, brown or black. The enamel is pitted and the teeth themselves become unduly brittle and soon deteriorate, while fillings or stoppings tend to fall out.

Degeneration of the skeleton in adults occurs when the level of fluoride is above about 6 p.p.m. and at the same time degeneration of the kidney may occur. The spine becomes converted into a bowed and solid column of bone, fused to the skull and to the ribs and to the pelvis, while the other bones degenerate and thicken, and the joints and ligaments become stiff and calcified. These bony changes cause pain by pressing on nerves; arthritis; stop expansion of the chest while breathing; and produce anaemia and deafness by encroaching on the blood-forming marrow and on the organs of hearing (41). Such ghastly results have never been seen in England where the levels of fluoride never reach those in some parts of Africa, India and China.

Fear that fluorides in drinking water even at the level of 1 p.p.m. may cause ill-health in children and even more in adults and the aged, has caused many people to protest at the deliberate addition of fluorides to water supplies in order to lessen dental decay. Indeed after two years of such an addition the people of Andover were about to bring a legal action against their Council for poisoning their water, but unfortunately the legal right of a Council to add whatever it likes to water was never vindicated as the Council itself stopped fluoridation on the 1st July 1958.

Now fluoride is only being added to water in Watford and in parts of Anglesey and in Kilmarnock. But such additions have been made in the U.S.A. for over ten years and the most meticulous and well-controlled examinations of children in fluoridation areas have shown no effects at all except the desired reduction in dental decay; and the death rates of adults in areas where there has always been a very high natural level of fluorine in the water are no different from the death rates in fluoride-free areas, nor is there any difference in the incidence of heart disease, kidney disease, cancer, cirrhosis of the liver, strokes, etc. English figures are

scanty but they support the American as regards deaths from kidney disease; a tentative early report (38) that the natural fluorides in the water at Bampton and Launton in Oxfordshire, and at Maldon in Essex, caused abnormalities in the spines of children has not been confirmed (42); that fluorides may play a part in causing simple goitre (p. 18) in children is possible (37), though it cannot be important since most investigators of children with mottled teeth have never noticed an increased incidence of goitre.

But while it is certain that the average child and adult will not be harmed by drinking water all their lives containing about 1 p.p.m. of fluoride, yet the exceptional individual may still suffer: an unusually high fluid intake, for instance, could well raise the amount of fluoride absorbed to a dangerous level—I have seen two utterly fit patients who both drank 30 pints daily, one being a stoker who, wanting money to marry, worked a double shift of 16 hours a day, while the other was a professional dancer. I have also seen a fit boy aged seven who always drank 6 pints of water daily—an amount which is common for children in hot weather. Again poor diets, and especially diets low in calcium or vitamin C, are said to enhance the absorption or toxicity of fluorides. Though in England there is, in theory, no grave malnutrition yet there must be an appreciable number of people who live on odd and fluoride-high diets such as tea, tinned pilchards and baking-powder breads. Again it is a worrying possibility that an individual may be unduly sensitive to fluorides, as is suggested by only a small number of children developing mottled teeth in a high-fluoride area; or, though statistically there is no support for it, that an illness may be accentuated by fluorides: that a sick kidney may find the burden of fluoride excretion overwhelming, that the arthritic spine of the old may be stiffer and more crippling when acted on by fluorides.

In other words mass medication can never take into account the exception. How far democracy, to benefit itself, is morally entitled to sacrifice the minority is a nice point, especially when the minority may possibly be so small as to be virtually non-existent.

Finally the decision whether all the waters in Great Britain are to be fluoridated or not will be solely decided, though this may startle the depthless, by the English, Scotch, Welsh and North Irish deciding not whether fluoridation is beneficial but whether it is morally right to force people to take fluorides against their wish.

Surely the answer, though far less satisfactory for decreasing dental decay, is not compulsory fluoridation of water which no one can avoid drinking, but the fluoridation of white sugar, sweets and soft drinks, those easily avoidable but universally consumed causes of dental decay.

CHAPTER XII

The Prevention of Congenital Abnormalities

'*He* (*Professor Platt*) *described the various far-reaching effects which might be caused by small amounts of impurities being present in food. It was possible that not enough care was taken in the consideration of the effects of the impurities which were added to food by accident, or deliberately. Not enough details were known about the action of these impurities. Substances like pesticides, insecticides, and flour improvers should be used with great caution until there was more knowledge about their effects. There was a possibility that they might be responsible for some disorders of the development of the brain*' (166)

'*Preservatives, Dyes and "Improvers" in foodstuffs, which are widely used with no recommendation other than that of being toxic to bacteria, or colourful or producing a white frothy bread are so widespread that, if any were to cause embryonic damage, it would be extremely difficult to detect. . . . Among factory-produced foodstuffs . . . may be mentioned the colouring matter in margarine, the large number of mixtures which are supplied as dehydrated cakes and pastries and ice cream*' (167)

'*In fact, it is likely that at least some of the cancers observed at birth or in infants and children are attributable to an exposure of the maternal organism before or during pregnancy or lactation, to carcinogenic agents which passed the placental barrier or were secreted in the milk*' (62).

The Prevention of Congenital Abnormalities

Congenital abnormalities, physical and mental, are mainly caused by the alien substances added to food.

Many theories, until now, have been held about the causes of congenital abnormalities: they are inherited; they are due to maternal infections during pregnancy; they are caused by physical accidents to the mother such as a fall; they are caused by a deficient maternal diet. But none of these can explain most of the observed facts about the families or the happenings during foetal life of children born headless, or with gross or minor defects of their central nervous system or of their eyes or hearing or hearts or lips or palate, etc. Heredity at best appears to be only a background factor: it may predispose a foetus to faulty development but a 'second hurtful factor' is needed to pervert foetal growth. Physical accidents during pregnancy certainly do not provide this 'second hurtful factor' nor is there any valid evidence that they can cause damage by themselves alone unless they are most violent. Maternal infections certainly may cause abnormalities, since it is definite that rubella, German measles, in some epidemics though not in all, may injure the foetal heart, eyes and hearing if the mother is infected—or even exposed to German measles without being apparently infected (169)—during the first three months of pregnancy, just when the heart, etc., is being formed. But only a small proportion of children born of infected mothers are malformed, which again suggests that German measles only affects the foetus when it is also being devastated by a 'second hurtful factor'.

In passing, it is comforting to emphasize that today syphilis virtually causes no abnormalities, nor, when it does, are these in the 'mysterious' group of anomalies discussed here, being caused by a definite infection of the infant itself.

Lastly is left the possibility that a deficiency in the maternal diet can injure the foetus in the same way that in

animals many abnormalities, apparently analogous to human abnormalities, can be produced by a deficiency, for instance, of vitamin A (83) But, in fact, there is no association between human deficient diets and foetal malformation, possibly because the human foetus is such an efficient parasite—essential in a species with very low fertility—that the mother dies before it is starved; or because experimental animal diets are generally perfect, apart from lacking the one essential nutrient being investigated, so that all the foetal tissues save those dependent on the missing nutrient grow normally and thus a maimed foetus survives, while in man a seriously deficient diet always lacks many essentials, so that either total foetal development is possible or none. This may not appear to be borne out by the beneficial effect of giving extra wheat germ oil during pregnancy, but this is capable of a better explanation, given below, than considering women are often deficient in the ingredients of this oil.

Thus we are left with none of the classical explanations of congenital deformities and with no explanation of why some are commoner in children born in the winter, some commoner in those born in the summer; some rarer, for instance, in the Scotch Highlands than in the Lowlands; some rarer on the Continent than in Great Britain; some much commoner in 'less well off' sections of the community, though these are 'rich' in the sense of being able to afford the foods they need or desire.

The toxic substances added to our foods explain the whole of this tangle. They are the 'second hurtful factor'. The faint effect of heredity is seen merely as reinforcing the injury caused by food additions; the different foetal toxicity of German measles during different epidemics reflects the local introduction or withdrawal of a particular kind of margarine, cake-mixture, insecticide, etc.; the strange seasonal variations are merely the result of normal seasonal

But all that this and similar reports (83) show is that the addition of essential constituents to the diet has prevented disaster in a large proportion of women. It well may be that they were deficient in vitamin E and essential fatty acids owing to eating chemically treated bread (p. 62) and margarine (p. 68) but equally well the good effects may have been due to vitamin E reinforcing the liver's capacity to detoxify the poisons in their food (174).

Parents, wives *and* husbands, if not for their own sakes then for the sakes of their children—unconceived, conceived, born—should avoid all foods containing alien substances.

CHAPTER XIII

The Prevention of Cancer

Prevention of most cancer is possible, better treatment unlikely. Prevention not treatment is the only way of reducing the rising mortality from cancer. Prevention is entirely in the hands of the individual: its essence is the avoidance of cancer-causing substances.

Most cancers are not due to endogenous causes, to causes arising within the body itself, but to exogenous causes, to insults to the body from external sources. This is why cancers generally arise in those parts of the body which are exposed to the influences of the outside world: the skin to X-rays, the lungs to smoke, the stomach and bowel to food chemicals, the kidneys and bladder to the chemicals absorbed from food which they concentrate and excrete.

In order to understand prevention it is necessary to consider the two peculiar properties of cancer-causing substances.

(1) The Latent or Incubation Period

Cancer does not develop until, perhaps, ten or twenty years after a man's last exposure to the substance causing his cancer (210) Thus of 82 men who developed cancer of the bladder as a result of working in a dye factory, one man had stopped work for 34 years, one for 33 years, nine for more than 15 years and nine for between 10 and 15 years.

neutralizing cancer-causing chemicals. Each time we eat such a chemical we spend some of our irreplaceable capital: when the capital is spent we die of cancer.

Therefore the individual cannot afford to eat anything which he thinks is cancer-causing. He must fight to leave himself all the margin of safety he can, so that he has used up none of his reserves before he is faced with some cancer-causing chemical which he will probably neither know exists nor be able to avoid, such as, possibly, some detergents and some eggs. Eggs are not 'food' until they have been laid by the hen. Therefore the pallid yolks of commercial battery eggs can legally be coloured with any yellow dye, however dangerous, if, being fed to the hen, it is excreted into the yolk. Thus deluding the public by providing battery eggs with yolks dyed to the golden yellow of the yolks of 'farm' eggs is a dangerous swindle and should be banned.

The demands must not be granted of food manufacturers that they should be allowed to continue adding minute amounts of cancer-causing chemicals to food in the same way that they are allowed to add ordinary poisons within prescribed limits. This idea of a tolerated maximum for noxious substances in food dates from the days when all food contaminants were simple poisons, so that the only problem of their control was to ensure that a dangerous dose was not left in food. Food manufacturers, having been brought up with this idea of a safe dose, naturally clamour for a safe dose to be declared for all substances they use. But the realization that the injury from cancer-causing chemicals is throughout life cumulative and never healed, leaves no logical safe alternative to the complete banning of every chemical which has ever caused cancer in any way whatsoever in animals or man (p. 43).

The particular cancers discussed until now have been those which obviously are or could be due to exogenous causes, to

causes external to the body such as chemicals in foods which directly impinge on the stomach and bowel or, after absorption, are concentrated in the urine and so affect the urinary tract. *Cancer in other organs of the body not directly exposed* to the external world might seem less explicable and less preventable in terms of food. But, at least in animals, cancer in any organ of the body may be caused by eating particular chemicals: for instance 2-acetylaminofluorene (p. 85), causes cancer of the external auditory canal, the eyelid, the tissues behind the eyes, the thyroid gland, skin, breast, lungs, liver, bladder, kidney, small and large intestine, pancreas, uterus, seminal vesical, and also of the pituitary gland if stilboestrol is taken at the same time. So however unrelated to food a cancer may appear to be, there can be no doubt that it may in fact be caused by chemicals in food. How far other factors like obesity (208), vitamins (83), worry (213), etc., reinforce or restrain the cancer-causing activity of food chemicals is a fascinating facet of cancer prevention which is not, unfortunately, germane here except for the role played by hormones in cancer of the breast.

Cancer of the breast, or at least one form of cancer of the breast occurring before the menopause, is caused by or is dependent for its development and growth on oestrogens, that is on the female sex hormones. These hormones are even capable of causing cancer of the breast in men (214) who have been treated with large doses to control cancer of the prostate—a queer example of a substance being able to suppress one form of cancer while causing another. In order to remove the stimulating effect of the oestrogens on cancer of the breast, such cancer is now often treated with surgery and X-rays combined with surgical removal of the woman's ovaries, that is removal of the glands which make oestrogens. But when the ovaries are removed the suprarenal glands may

CHAPTER XIV

The Prevention of Diseases of the Arteries, Heart and Kidneys

Arterial degeneration is the cause of arteriosclerotic heart disease, angina and coronary thrombosis; of cerebral haemorrhages, thromboses or 'strokes'; of some renal diseases. But it is the insidious degeneration of the cerebral arteries with its insidious destruction of the mind that causes most unhappiness—that causes the interminable mental illness which is old age.

A high level of cholesterol in the blood—hypercholesterolaemia—is now considered, on very convincing evidence from man and animals, to be the essential cause of arterial degeneration. This excessive cholesterol invades the arterial wall, destroys its structure and forms plaques on its inner surface: these plaques reduce the diameter of the artery so that, as in angina pectoris, too little blood can flow through; or, as in coronary and cerebral thrombosis, blood clots on their rough surface and the artery is completely blocked.

Cholesterol is a substance closely akin to vitamin D, to the sex hormones, to the hormones of the suprarenal cortex and to the cancer-causing hydrocarbons—it is, indeed, cancer-causing itself in particular circumstances (195). A diet rich in solid animal fats or in hydrogenated oils like margarine (p. 68) is one of the main causes of a high level of cholesterol in the blood, while the liquid vegetable oils

containing essential fatty acids or E.F.A. (p. 69) reverse the high level caused by the solid fats.

Arterial degeneration is increasing rapidly in all civilized countries: is, indeed, a disease of modern civilization. And since arterial degeneration and food sophistication are progressing hand in hand it would seem probable that the cause of this degeneration, or, rather, the cause of the antecedent high level of cholesterol in the blood, is not the excessive consumption of normal food, as has been suggested, but the excessive consumption of abnormal food.

For our diet before the war and today is almost the same in its major ingredients (196), apart from milk consumption rising from 174 to 265 pints per head annually and margarine rising from 9 to 17 lb. with a parallel fall in butter from 25 to 16 lb. If, therefore, our increasing arterial degeneration is caused by our present food, we must either incriminate the new chemicals in cakes, bread, meat, vegetables, etc., or those foods of which more are eaten. Whether or no chemicals such as emulsifying agents, artificial colours, agricultural insecticides, etc., damage the arteries is unknown and little help is given from experiments on animals since these are largely immune to arterial decay on their usual laboratory diets. But it seems reasonable to believe that incessant trivial damage is done to the arterial lining by any often-eaten toxic substance, and that such damage may fail to be repaired but rather open the way for progressive arterial degeneration from the hypercholesterolaemia caused by the increased consumption of perverted ingredients of the diet such as margarine and milk.

Margarine, including other hydrogenated vegetable oils like the 'cooking' fats or 'frying' fats (p. 68), is particularly suspect: arterial degeneration and an increase in margarine consumption have gone forward together in Great Britain, while the inverse happened in Norway during the war: the

for their condition and at autopsy had two to three times as much lead in their bones as patients who had had some reason, apart from lead poisoning, for their nephritis.

Lead is not the only poison to which children or young animals are highly susceptible, so it would seem especially urgent to protect children against all accidentally contaminated foods which may have no overt effect on their kidneys until many years later.

CHAPTER XV

The Prevention of Diseases of the Nervous System

There are a large number of degenerative diseases of the nervous system all with no known cause. This unknown cause is most unlikely to be the same for each disease because each differs so widely from the others. So it would be absurd to suggest that all, or indeed any neurological diseases are entirely due to injurious substances in food. On the other hand it would seem reasonable to consider whether some abnormalities in foods may not accentuate or worsen diseases after these have appeared, or, indeed, to an apparently normal nervous system be the final insult which unmasks its weakness and precipitates its collapse. In other words abnormal foods plus other damaging influences all add up to a combination of strains which the nervous system cannot withstand

But this is, of course, essentially a theory which is not provable: a patient who has already developed a nervous disease may indeed appear to be better by avoiding margarine, etc., but then he might have been better in any case; a man whose family history suggests he is prone to a nervous disease may well remain unaffected whether or no he ponders on his diet. Probably the best way of assessing the wisdom of paying attention to food as a treatment or as a preventative is to consider how far food can explain the

known facts which are puzzling about neurological disorders. The commonest and most widely investigated disease being disseminated sclerosis, full consideration of this in relation to food will show in general how far foods may be an important partial cause in this and other diseases, the latter being separately mentioned later if they have any particular relation to certain food poisons.

Disseminated Sclerosis in Great Britain affects about one in two thousand people. In essence it consists of multiple patches of inflammation scattered anywhere throughout the brain and spinal cord, which lead to demyelination—the stripping from the nerve fibres of their insulating fatty sheath of myelin. Hence the name 'demyelinating diseases' is sometimes given to disseminated sclerosis and to possibly kindred conditions such as acute disseminated encephalomyelitis and neuromyelitis optica. Myelination of the nerve fibres starts about the fourth month of foetal life and is complete before the age of two years.

Myelin is built of many substances including the essential unsaturated fatty acids (p. 69) which the body cannot make itself but has to obtain ready made from food. So it is important that during pregnancy and in the years after birth the diet should contain no abnormal or spurious fatty acids, such as those in margarine (p. 69), which being so similar to the essential fatty acids may delude the body into using them to build myelin. For it is reasonable to suppose that myelin built of counterfeit material will be pinchbeck and liable to disintegrate under strains withstood by normal myelin.

Once myelination is complete it is unknown whether there is any change or turnover of its ingredients and whether, if it is damaged, it can be repaired. It would seem inherently likely that at least repair is possible. So that throughout life, and certainly throughout the life of anyone with a demyelinating disease, it is unwise to eat shoddy building

stones—margarine, cooking fats, our usual bread and flour.

The unwisdom of eating margarine is shown in Norway: during the last war the Germans destroyed the margarine factories so that there was virtually no margarine. But in the two years after the war three times as much margarine was eaten as had been eaten before the war, and at the same time there was two and a half times as high an incidence of disseminated sclerosis as there was during the margarine-free war period (193).

The facts about disseminated sclerosis, which any theory about its cause must explain, are the slight but definite hereditary or familial incidence; the different incidence in different countries, South Africans of any colour or race being virtually immune if they have *never* been away from their country, the patchy incidence in the same country, it being more than three times as great in Shetland as in the Western Isles and Skye, and inland Norwegian farmers are affected more than fishermen on the coast; the erratic progress of the disease.

A family history of disseminated sclerosis is found in 5 to 10 per cent of patients, but it is their brothers and sisters rather than their parents or their children who are also affected. In other words if there is an inherited tendency toward disseminated sclerosis it is very slight and tends only to be unmasked in one particular generation. Such a generation both during foetal life and childhood has had the same diet, so it is arguable that it is the damage done by injurious food during the formation and growth of the nervous system which later in life makes an hereditarily weak system collapse. It is even possible that there is in fact no genetic inheritance of disseminated sclerosis: even when several different generations are affected they are not suffering from any inherited weakness but merely from a long family tradition of feeding.

Such a casting away of all inherited genetic factors certainly makes the whole problem easier. The perplexing fact that South Africans who visit Europe and then return home may many years later develop disseminated sclerosis, while those who have always stayed at home do not, can be simply explained by the travellers having eaten some food, not available in their own country, which has damaged or been built into and thus weakened their nervous system so that it fails to withstand normal stresses encountered years later.

Lead and copper also fit into a dietetic theory. There is evidence that a high level of lead in the soil predisposes to disseminated sclerosis (*169*, *192*) and that the teeth of patients contain an unusually large amount of lead (192). Lead alone does not cause disseminated sclerosis or it would be pandemic among lead workers, but its presence in the soil may interfere with or pervert some other substances present in vegetables, etc., grown on lead-rich soil. This possibly far-fetched idea is supported by the prevention of 'swayback' in lambs. This is a demyelinating disease of new-born lambs which can be prevented by feeding the ewes extra copper (194). Swayback, however, occurs on certain pastures which are not deficient in copper, so it appears that some injurious factor in certain pastures needs extra copper to neutralize it. In a similar but opposite manner lead may neutralize the beneficial action of copper or some other substance.

The patchy distribution of disseminated sclerosis, whether it is associated with certain cottages in a village or with farming as against fishing activities, again can be seen as being caused by excess of lead, etc., in the soil of the cottages or by traditional variations between the food of farmers and fishermen.

The erratic course of disseminated sclerosis may be due partly to the amount of damage already done to the nervous

system by foods, so that unnoticed stress may cause a breakdown or even severe strain may be survived; partly on whether good or valueless building materials for patching the damaged nervous system are being eaten.

Residues of organo-phosphorus insecticides (p. 89) left on foods are the best example of food 'stresses' which might cause or precipitate demyelinating disease. Permanent demyelinating paralysis has already been caused in man through working with one of these insecticides (p. 92) and some 15,000 people were paralysed through drinking 'Jamaica Ginger' which was adulterated with a demyelinating chemical having some physiological relation to the insecticides (p. 92). Other chemicals are also known which cause demyelination in some animals. But what is particularly disturbing is that these chemicals tend only to destroy the nervous systems of birds and of man, but not the nervous systems of the animals commonly used to test the safety of chemicals added deliberately to or accidentally left in foods. So there is even less protection from animals experiments than usual against foods being contaminated with demyelinating substances.

Parkinsonism, Paralysis Agitans or *The Shaking Palsy* is a common disease following on degeneration of the nerve cells of certain lower cerebral centres. Sometimes the patient gives a history of an acute virus encephalitis, but generally no cause for the degeneration can be found. It certainly cannot be ascribed to any modern food additions or contaminants since Parkinson gave his description of the illness in 1817; nor have any chemicals produced in man or in animals exactly comparable degenerations. But even so it would seem wise for patients to avoid foods possibly contaminated with any of the chemicals which damage the nervous system—lead, mercury, many of the agricultural insecticides (p. 91) and, indirectly, the emulsifiers used in commercial cooking,

and crustaceans'. Among animals killed were foxes, armadillos, squirrels, mice and in some places rabbits were 'decimated or wiped out'. In a residential area 36 homes reported the deaths of 697 chickens, 20 turkeys, 11 cats, 2 ducks and 'upwards of 50 dogs were hauled to the city dump' (225).

Changing the flora of the countryside by wantonly destroying the plants in hedges or road verges with herbicides, or by ploughing up moors, etc., for crops will of course affect the fauna which live on particular plants: they migrate or starve or even are poisoned. The oddest example of the last comes from Australia where grass pastures were ploughed up and reseeded only with Subterranean Clover (*Trifolium subterraneum*). The resulting virtually pure clover pasture did not harm grazing cattle, horses, or rams, but ewes became infertile with dystocia, prolapse and inversion of the uterus; virgin ewes and castrated lambs lactated (270). This was all due to the clover naturally containing a large amount of oestrogens.

Birds

Dying birds hide themselves and are also killed by their enemies, so it is impossible to know how many birds are killed by agricultural chemicals. Again, when large numbers of, for instance, poisoned thrushes are found dead together only one or two will be sent to a laboratory to decide the cause of death. Therefore laboratory figures underestimate the role poisons play in bird mortality.

Of 460 dead wild birds examined in one laboratory (228), over one-third had been killed by agricultural poisons. The birds killed were wood pigeons, pheasants, chaffinches, rooks, stock doves, blackbirds, house sparrows, partridges, feral pigeons, jackdaws, coal tits, skylarks, song thrushes, greenfinches, bramblings, mute swans, black-headed gulls,

greater black-backed gulls and herring gulls. Owls have been killed by eating rats poisoned with phosphorus (229), and many dead pigeons have recently been found each containing enough nicotine to kill two men (230).

Cuckoos have not been reported as being poisoned in England, but their absence in East Africa on their normal migratory routes has been ascribed to their poisoning by insecticides or to the insects on which they live being poisoned (231).

How birds become poisoned is of great importance. When 158 dead birds are found in 46½ acres of Brussels sprouts just after spraying (p. 137), it seems obvious that they were directly and rapidly killed by the poison itself falling on them or being breathed in in the spray. But what is far more important is the rendering poisonous of the insects, worms, etc., which are the bird's food. This so prolongs the poisoning of the birds that they may die far from the sprayed crop and months later, so that there is no discernible relation between their death and the spraying. Further, their eggs may be infertile or the young weak (232), causing a further unrealized mortality.

When elm trees are sprayed in July against elm disease and again during the winter, the immediate death of birds may not be great. But the DDT remains on the July leaves, and remains in the leaves when they fall in the autumn and rot. The rotten leaves are then eaten by worms. These worms are eaten in the spring by robins and other birds. The birds die (232, 233).

Again, the winter spraying makes poisonous the insects which live in the bark of the trees. In the winter and spring these too kill the engaging little tree-climbing birds—woodpeckers, creepers, nuthatches, etc.

Again, the birds of prey, hawks and owls, are killed by eating birds made poisonous by eating worms made

poisonous by eating sprayed elm leaves. The chain of poisoning goes on and on—do some chains ultimately complete the circle and come back to man?

Of course all spraying of trees in orchards, etc. and all spraying of any plant leaves which subsequently fall to the ground causes the same lethality of worms and insects in the spring.

But there is no value in recording more and more accounts of birds decimated or wiped out (232) by agricultural poisons, except perhaps to emphasize that even seed corn dressed with dieldrin has killed hundreds of pheasants and other birds after it has been sown and then eaten (234): that even wood-preservatives like the chlorinated naphthalenes (p. 88), when used on nesting boxes can be lethal. No commercial nesting boxes should ever be used in gardens, woods, etc. They should be home made (272) of thick unpreserved, unpainted, untreated wood. They should face north (272).

'The current widespread and expanding pesticide programme poses the greatest threat that animal life in North America has ever faced—worse than deforestation, worse than market hunting and illegal shooting, worse than drainage, drought, oil pollution, and possibly worse than all these decimating factors combined—we shall have been witnesses, within a single decade, to a greater extermination of animal life than in all the previous years of man's history on earth. . . .' (232). Admittedly this was written by an American, but pesticides and their effects, like science and inhumanity, are international.

Bees and Insects

Bees collect each year honey worth more than £1,000,000 and we spend another £700,000 on imported honey: the value of our own beeswax is unknown but imports cost

about £500,000, being chiefly used for cosmetics and for polishing the lenses of lighthouses.

The true importance of the bee, however, is as a pollinator: everyone knows that beehives may be especially brought to orchards, or to crops of clover grown for seed, in order to pollinate the flowers and so increase the yield of fruit and of seed—indeed the acreage used for clover seed could be reduced by 30 per cent if all farms had ample bees (246). But even the agricultural community appears not to realize that remedying the modern dearth of bees increases by about 2½ times the yield of swede turnip seed, kale seed and black currants (247) and the yield of beans (248), while lavender oil is increased by 16 to 20 per cent (249). The once flourishing flax growing industry of Warwickshire has been wiped out by lack of bees (250). Even the yields of self-pollinating plants like apricots and pears and wheat are said to be greatly increased when bees are plentiful (251). Bees avidly attracted to flowers show that the plants are not receiving too much nitrogen for seed formation (258).

There are not ample bees in England in spite of the repeated statements by Ministers in Parliament: were there ample bees no such increase in beans, black currants, etc., would occur when beehives are imported into the fields. By definition, ample bees means enough bees to cause the largest possible crops.

Insecticides are largely to blame for this famine of bees, which is not surprising as bees are insects. Proof that bees have been killed by insecticides used on one particular farm is difficult, proof to the extent that the bee keeper can obtain damages from the insecticide sprayer is generally impossible, since bees range over about 12 square miles round their hives and in so large an area many farmers may have used insecticidal sprays and so no one can tell which farmer killed the bees. But even so it is clear that agricultural

poisons are the reason why, for instance, beekeepers in Cambridgeshire have dwindled from several thousand to a few hundred, while bee journals have recurrent accounts of apiaries virtually wiped out through orchards and field crops in bloom being sprayed—indeed one distributor of insecticides admitted he had destroyed 50 colonies of bees because he 'had to' spray a crop in bloom (252). While the farmer may have benefited from the spray, the beekeeper lost about £1,000 worth of honey and his bees.

The chemicals most lethal to bees are DDT, BHC, Chlordane, DNC, parathion, and arsenic (221); and lindane (253); and dieldrin (227); while DNOC is less toxic (254).

Honey is unlikely to be contaminated with the insecticides most toxic to bees because they will die before their honey is stored in the comb, but the systemic insecticide schradan (p. 89) applied to plants *before* they flower appears in their nectar and, being non-toxic to bees, is then stored but not decomposed in the honey, which, according to the only work done on this important subject (256), may contain as much as 44·5 p.p.m. of schradan. This would equal 20 mg. of schradan in one pound of honey, an amount eaten by many people in a few days. 'This aspect of food contamination must be given careful consideration. These values may by no means present maximum values and might be different in other crops' (256), or with other systemic insecticides (255).

Other insects are killed in the same way as are bees. The drawbacks of this destruction are: pollinating insects such as bumble bees, wild bees and pollen beetles cannot redress the scarcity of domestic bees; the beneficial insects—parasites or predators—being killed, obnoxious species such as the red spider mite multiply greatly to fill the 'biological vacuum' left after heavy repeated spraying; the insect food of animals and birds and fish is destroyed or rendered

poisonous; pleasing insects like butterflies fade from the landscape.

Earth Worms and Soil Organisms

In an acre of healthy grassland there should be one to three million worms (273): the weight of worms under the pasture should equal the weight of the grazing animals on the pasture. Worms every year throw up 8 to 16 tons of worm casts per acre (273, 274)—from a depth of up to 8 ft.—which are far richer than the surface soil in both inorganic and organic plant nutrients. Worms also aerate, break up and drain heavy soils, and draw down into the soil soil foods such as dead leaves, etc.: in fact they are tireless unpaid ploughmen and fertilizers.

But worms are starved or poisoned by artificial manures and agricultural insecticides, etc. For instance the insecticide heptachlor is reported to have reduced—after one application—the earthworm population by 79 per cent (225) and, 'A visit to almost any orchard in Kent during the spring immediately after the trees are sprayed with tar oils or lime sulphur will be sufficient to prove how harmful this spraying is to the earthworm population. The ground soon afterwards is covered with a carpet of dead worms' (259).

Worms rendered poisonous by insecticide may be lethal to birds many months after the spraying is over; robins, for instance, being killed in the spring from eating worms containing chemicals sprayed the previous summer (p. 139); woodcock have died from eating worms in areas sprayed five months before (225). Fish, also, are killed by eating DDT poisoned worms washed down streams after spraying (224).

Other inhabitants of the soil are as important as earthworms. No clear cut work appears to have been done on the effects of insecticides, etc., on these organisms but the

destruction, for instance, of the predator of the eel worm by chemical farming emphasizes how dangerous it is to ignore the covert effects of chemicals.

Fish and Aquatic Life

'An acre of water is worth far more than an acre of average land.... In China, Germany, Austria, Hungary and Poland the average harvest of carp is two tons per acre' (235).

In the Ymsen lake in Sweden, only 5 miles long by 2 miles at its broadest and 6 to 8 ft. deep, over 44,000 pounds of various kinds of fish are caught annually (236). In an English Wiltshire lake of 30 acres scores of thousands of fish were caught when it was last netted, though the present owner does not even know what kinds of fish are in his lake. In fact all over England there is a complete lack of interest in fresh water fish as food, apart from salmon and trout, though carp and pike and eels are preferable to any of the fish, except salmon, sold by up-country fishmongers.

But this lack of interest is no reason for ignoring the effects of agricultural poisons on fish: angling gives pleasure to about a million fishermen a year and would to more were there less destruction of fish by polluted water; further, fish could be one of the pleasanter sources of first-class protein. To destroy them when they are a source of food which is completely under our own control seems folly, especially as half of our food is beyond our own control, depending on the surpluses of other countries. The extravagance and the precariousness of this dependency are well illustrated by salmon. Ten million pounds are spent annually on imported tinned salmon, but if all the rivers in England were protected from pollution *fresh* English salmon would sell in season at 2s. to 3s. a pound (237). Even the supply of im-

ported tinned salmon is precarious: Canada, our best supplier, is now spraying DDT from the air on the forests along her main salmon rivers and 'almost immediately alarming quantities of dead fish were found floating near the mouths of some of eastern Canada's most famous salmon rivers'. Further, in spite of the greatest precaution in spraying, 90 to 95 per cent of salmon fry placed in pens in the river were killed and a vast quantity of the aquatic insects on which they feed were also wiped out. Such spraying, combined with hydro-electric dams of the type now being built in England, will reduce the salmon-runs to 'economic extinction' (238), while we in England will have no tinned salmon and, having killed our own fish, no fresh salmon either.

Though the immediate effects of agricultural sprays, etc., are often obvious they are difficult to prove legally and legally to prevent. The fish die, are washed away and decompose, so analysing them to show they died of an insecticide, etc., is generally impossible though the cause of their death is obvious. Thus about 3,000 beautifully conditioned trout were killed suddenly in a five mile stretch of the Pinsley Brook in Herefordshire. But the verdict on this slaughter, fully investigated, was only, 'We suspect it was a chemical used for spraying crops' (239). Again, near Spalding in Lincolnshire, roach, rudd, bream, pike and perch to the value of £1,000 were found dead (240) but it could only be 'thought to be' due to agricultural insecticides. But in the Blue Nile (241) it was conclusively proved that DDT sprayed on the river to give a local concentration of only 0·09 parts per million caused 8 miles lower down, where it must have been greatly diluted, the deaths of hundreds of fish—both plant feeders and carnivorous bottom feeders: the fish concentrated the DDT, up to 79 p.p.m. being found in their viscera and 64 p.p.m. in their fat. In the U.S.A. (232) up to

Appendix

Dyes

The dyes which are used or have recently been used in food are predominantly azo dyes, containing the group:

$$-N=N-$$

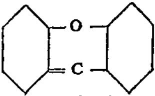
(Azo)

while other less common dyes have the following groups:

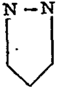
$$-NO_2$$

(Nitro)

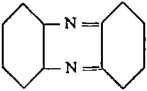
$=C(C_6H_5)_2$ (Diphenylmethane) $-C(C_6H_5)_3$ (Triphenylmethane)



(Xanthene)

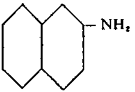


(Pyrazolone)

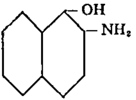


(Azine)

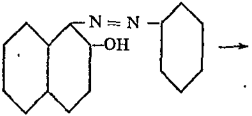
Dyes with the nitro or azo groups are very suspect because in many cases they can, or could in theory, be converted in the body into an aromatic amine with the position *para* to the amino-group blocked by a large substituent, so that subsequently the cancer-producing *ortho*-hydroxy amine group is formed. A simple example is the formation in the body from 2-napthylamine:



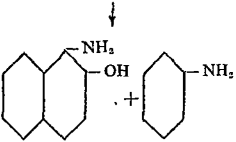
of the cancer-producing *ortho*-hydroxyamine:



or the formation, presumably, in the body of *ortho*-hydroxyamine from the azo dye Sudan 1, which produces cancer in animals and was in the 1940's widely used to colour margarine. Thus:

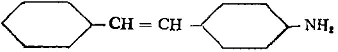


Sudan 1,



(*Ortho*-hydroxyamine)

The colourless dye which may be used in domestic detergents is related to the cancer-causing:



(4–Aminostilbene)

Detergents: Surface-Acting Agents

The most common surface-acting agents in domestic detergents are of the alkylarylsulphonate type:

$$R.C_6H_4.SO_3Na$$

R = an alkyl radicle containing 9 to 15 carbon atoms.

Less widely used are the alkyl sulphates:

$$\begin{matrix}R_1\\R_2\end{matrix}\!\!>CH.OSO_3Na$$

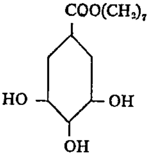
R = a higher alkyl radicle and R_2 a higher or lower alkyl radicle.

Appendix

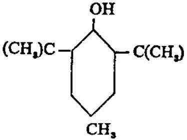
The following are the only coal tar colours now permitted in foods in England.

Common Name of Colour	*Scientific Name*	*Colour Index (1924) Number*
Ponceau MX	disodium salt of 1-(2.4-or mixed-xylylazo)-2-naphthol-3:6-disulphonic acid	79
Ponceau 4R	trisodium salt of 1-(4-sulpho-1-naphthylazo)-2-naphthol-6·8-disulphonic acid	185
Carmoisine	disodium salt of 2-(4-sulpho-1-naphthylazo)-1-naphthol-4-sulphonic acid	179
Amaranth	trisodium salt of 1-(4-sulpho-1-naphthylazo)-2-naphthol-3·6-disulphonic acid	184
Red 10B	disodium salt of 8-amino-2-phenylazo-1-naphthol-3:6-disulphonic acid	30
Erythrosine BS	disodium or dipotassium salt of 2·4:5:7-tetraiodofluorescein	773
Red 2G	disodium salt of 8-acetamido-2-phenylazo-1-naphthol-3:6-disulphonic acid	31
Red 6B	disodium salt of 8-acetamido-2-*p*-acetamidophenylazo - 1-naphthol - 3.6 - disulphonic acid	57
Red FB	disodium salt of 2-[4-(1-hydroxy-4-sulpho-2-naphthylazo)-3-sulphophenyl]-6-methylbenzothiazole	225
Ponceau SX	disodium salt of 2-(5-sulpho-2:4-xylylazo)-1-naphthol-4-sulphonic acid	—
Ponceau 3R	disodium salt of 1-*pseudo*cumylazo-2-naphthol-3·6-disulphonic acid	80
Fast Red E	disodium salt of 1-(4-sulpho-1-naphthylazo)-2-hydroxynaphthalene-6-sulphonic acid	182
Orange G	disodium salt of 1-phenylazo-2-naphthol-6:8-disulphonic acid	27
Orange RN	sodium salt of 1-phenylazo-2-naphthol-6-sulphonic acid	26
Oil Yellow GG	a mixture of 4-phenylazoresorcinol and 4·6-di(phenylazo)resorcinol	23
Tartrazine	trisodium salt of 5-hydroxy-1-*p*-sulphophenyl-4-*p*-sulphophenylazopyrazole-3-carboxylic acid	640

Common Name of Colour	Scientific Name	Colour Index (1924) Number
Naphthol Yellow S	disodium or dipotassium salt of 2:4-dinitro-1-naphthol-7-sulphonic acid	10
Yellow 2G	disodium salt of 1-(2:5-dichloro-4-sulphophenyl)-5-hydroxy-3-methyl-4-*p*-sulphophenylazopyrazole	639
Yellow RFS	*disodium salt of 4-sulpho-4′-(sulphomethyl-*amino) azobenzene	—
Yellow RY	disodium salt of 6-*p*-sulphophenylazoresorcinol-4-sulphonic acid	—
Sunset Yellow FCF	disodium salt of 1-*p*-sulphophenylazo-2-naphthol-6-sulphonic acid	—
Oil Yellow XP	3-methyl-1-phenyl-4-(2:4-xylylazo)-5-pyrazolone	—
Green S	sodium salt of di-(*p*-dimethylaminophenyl)-2-hydroxy-3:6-disulphonaphthyl-methanol anhydride	737
Blue VRS	sodium salt of 4.4′-di(diethylamino)-4″:6″ disulphotriphenylmethanol anhydride	672
Indigo Carmine	disodium salt of indigotin-5.5′-disulphonic acid	1180
Violet BNP	sodium salt of 4:4′-di(dimethylamino)-4″-di-(*p*-sulphobenzylamino) triphenyl-methanol anhydride	—
Brown FK	a mixture consisting essentially of the disodium salt of 1:3-diamino-4:6-di(*p*-sulphophenylazo) benzene and the sodium salt of 2:4-diamino-5-(*p*-sulphophenyl-azo)-toluene	—
Chocolate Brown FB	the product of coupling diazotised naph-thionic acid with a mixture (Colour Index No. 1232) of morin and maclurin	—
Chocolate Brown HT	disodium salt of 2.4 dihydroxy-3:5-di-(4-sulpho-1-naphthylazo)benzyl alcohol	—
Black PN	tetrasodium salt of 8-acetamido-2-(7-*sulpho-4-p-sulphophenylazo-1-naphthyl-*azo)-1-naphthol-3:5-disulphonic acid	—

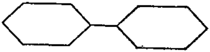


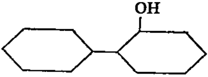
(Octyl Gallate)



(Butylated Hydroxytoluene)

Preservatives


(Diphenyl)



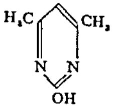
(Ortho-Phenylphenol)

$CS(NH_2)_2$
(Thiourea)

Coccidiostats



(DNC)

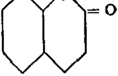


(HDP)

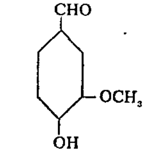
Nicarbazin is a molecular complex of both of the above.

Flavours

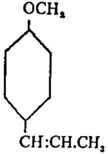
$(CH_3)_2C{:}CH.CH_2.CH_2.C(CH_3).CH.CHO$
(Citral: Lemon Flavour)



(Coumarin: Bad Vanilla)



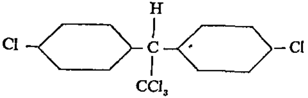
(Vanillin: Vanilla)



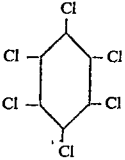
(Anethole: Aniseed)

$CH_3CO.CO.CH_3$ (Diacetyl: Butter)

Chlorinated-Hydrocarbon Insecticides



(DDT)



(BHC or Benzene Hexachloride)

Commercial BHC is a mixture of the four isomers, containing about 12 per cent of Lindane, the gamma isomer, which is the only insecticidal isomer.

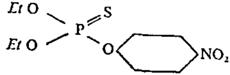
Organo-Phosphorus Insecticides

$$(Me_2N)_2\overset{\overset{\displaystyle O}{\|}}{P} - O - \overset{\overset{\displaystyle O}{\|}}{P}(NMe_2)_2$$

(Schradan or OMPA)

$$(Me_2N)_2\overset{\overset{\displaystyle O}{\|}}{P}F$$

(Dimefox)

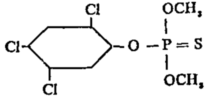


(Parathion)

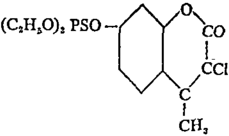
$$(iPrNH)_2\overset{\overset{\displaystyle O}{\|}}{P}F$$

(Mipafox or 'Isopestox')

Systemic Animal Insecticides.

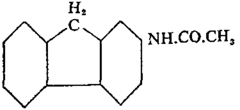


('Etrolene')



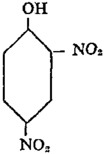
('Co-ral')

Cancer-Causing Insecticides No Longer Used

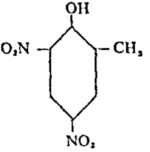


(2-Acetaminofluorene)

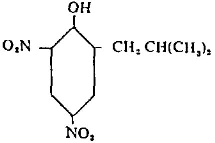
Dinitrophenol-Derivative Insecticides



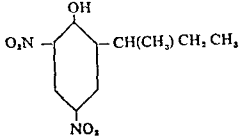
(2:4 Dinitrophenol)



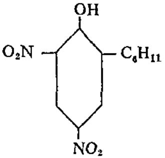
(2-methyl-4:6 dinitrophenol or 4:6-dinitro-*ortho*-cresol or DNC or DNOC)



(2-*sec*-butyl-4:6-dinitrophenol or Dinoseb or DNPB)

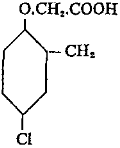


(2-(1 methylbutyl)-4:6-dinitrophenol or Dinosam or DNAP)

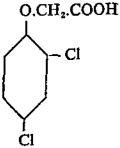


(2-cyclohexyl-4:6-dinitrophenol)

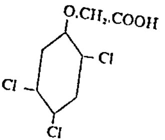
Herbicides



(MCPA or MCP)

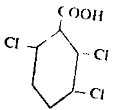


(2,4-D)



(2,4,5-T)

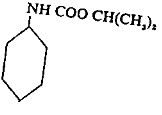
The first and last of the above, when the propri
radicle is substituted for the acetic acid radicle, for
prop or CMPP and 2,4,5-TP, while substituting the
acid radicle forms MCPB, 2,4-DB and 2,4,5-TB
herbicides are made by substituting ethyl sulphate,



(TBA)

$CCl_3\ COOH$
(TCA)

$CH_3\ CCl_2\ COONa$
(Dalapon)



(Propham)

References

(1) Mottram, V. H. and Graham, G., *Hutchison's Food and Principles of Dietetics*. 11th Ed., London, 1956, p. 461.

(2) Clements, F. W. and Wishart, J. W., 'A Thyroid-Blocking Agent in the Etiology of Endemic Goitre'. *Metabolism*, 1956, 5, 623.

(3) Morgans, M. E. and Trotter, W. R., 'Seasonal Variations in Thyrotoxicosis'. *Lancet*, 1949, 2, 1083.

(4) Savage, W. G. *Food Poisoning and Food Infections*. Cambridge, 1920, p. 194.

(5) Rouse, W. H. D., *The March Up Country. A Translation of Xenophon's Anabasis into Plain English*. London, 1947, p. 128.

(6) Pulewka, P., 'Über Andromedotoxinhaltigen Honig und eine biologisoce Methode zur Registrierung seiner Giftigkeit'. *Bull. Fac. Med. Istanbul*, 1949, 12, 274.

(7) Sergent, E., 'Les Cailles Empoisonneuse dans la Bible et en Algérie de nos Jours'. *Arch. l'Instit. d'Algérie*, 1941, 19, 161.

(8) Sergent, E., 'Les Cailles Empoisonneuses en France'. *Ibid.*, 1948, 26, 249.

(9) Bluck, R. S., *Plato's Phaedo*. London, 1955, p. 142.

(10) Chisolm, C., 'On the Poison of Fish'. *Edinburgh Med. Surg. J.*, 1808, 4, 393.

(11) Dodgson, R. W., *Report on Mussel Purification*. London, 1928.

(12) Gemmill, J. S., 'Mussel Poisoning'. *Med. Officer*, 1957, 98, 111.

(13) Meyer, K. F., *et al*, 'Mussel Poisoning'. *J. Prev. Med.*, 1928, 2, 365.

(14) Sommer, H. *et al*, 'Relation of Paralytic Shell Fish Poison to certain Plankton Organisms of the Genus Gonyaulax'. *Arch. Path.*, 1937, 24, 537.

(15) Crumpe, F., 'Observations on the Musculus Venosus and its use in Tetanus'. *Dublin J. Med. Sci.*, 1872, 54, 257.

(16) Casey, E., 'A Case of Oyster Poisoning'. *Brit. Med. J.*, 1894, 1, 463.

(17) Dack, G. M., *Food Poisoning*. 3rd Ed., Chicago, 1956, p. 31.

(18) Thesen, J., 'Studien über die paralytische Form von Vergiftung durch Muscheln (Mytilus edulis L.)'. *Arch. Exper. Path. Pharmakol*, 1902, 47, 311.

(19) McCarrison, R., 'Studies on Goitre Produced by Cabbage'. *Indian J. Med Res.*, 1931, 18, 1311.

(20) Cadart, J., *Les Escargots*. Paris, 1955, p. 75.

(21) Bodenheimer, F. S , *Insects as Human Food*. The Hague, 1951, pp. 55, 271, 281.

(22) Brunton, T. L., *A Text-Book of Pharmacology, Therapeutics and Materia Medica*. 3rd Ed., London, 1893, p. 957.

(23) Coxe, J. R., 'On Opium'. *Tr. Amer. Philosoph. Soc.*, 1794, 4, 387.

(24) Salaman, R. N., *The History and Social Influence of the Potato*. Cambridge, 1949, p. 124.

(25) Harris, F. W., and Cockburn, T., 'Alleged Poisoning by Potatoes'. *Analyst*, 1918, 43, 133.

(26) Tanner, F. W., and Tanner, Louise P., *Food-Borne Infections and Intoxications*. Champaign, Illinois, 1953, p. 152.

(12) Gemmill, J. S., 'Mussel Poisoning'. *Med. Officer*, 1957, 98, 111.

(13) Meyer, K. F., *et al*, 'Mussel Poisoning'. *J. Prev. Med.*, 1928, 2, 365.

(14) Sommer, H. *et al*, 'Relation of Paralytic Shell Fish Poison to certain Plankton Organisms of the Genus Gonyaulax'. *Arch. Path.*, 1937, 24, 537.

(15) Crumpe, F., 'Observations on the Musculus Venosus and its use in Tetanus'. *Dublin J. Med. Sci.*, 1872, 54, 257.

(16) Casey, E., 'A Case of Oyster Poisoning'. *Brit. Med. J.*, 1894, 1, 463.

(17) Dack, G. M., *Food Poisoning*. 3rd Ed., Chicago, 1956, p. 31.

(18) Thesen, J., 'Studien über die paralytische Form von Vergiftung durch Muscheln (Mytilus edulis L.)'. *Arch. Exper. Path. Pharmakol*, 1902, 47, 311.

(19) McCarrison, R., 'Studies on Goitre Produced by Cabbage'. *Indian J. Med. Res.*, 1931, 18, 1311.

(20) Cadart, J., *Les Escargots*. Paris, 1955, p. 75.

(21) Bodenheimer, F. S., *Insects as Human Food.* The Hague, 1951, pp. 55, 271, 281.

(22) Brunton, T. L., *A Text-Book of Pharmacology, Therapeutics and Materia Medica.* 3rd Ed., London, 1893, p. 957.

(23) Coxe, J. R., 'On Opium'. *Tr. Amer. Philosoph. Soc.*, 1794, 4, 387.

(24) Salaman, R. N., *The History and Social Influence of the Potato.* Cambridge, 1949, p. 124.

(25) Harris, F. W., and Cockburn, T., 'Alleged Poisoning by Potatoes'. *Analyst*, 1918, 43, 133.

(26) Tanner, F. W., and Tanner, Louise P., *Food-Borne Infections and Intoxications.* Champaign, Illinois, 1953, p. 152.

(55) *Annual Report of the Chief Medical Officer of the Ministry of Health for the year 1932.* H.M.S.O., London, 1933, p. 143.

(56) Monier-Williams, G. W., 'Aluminium in Food'. *Reports on Public Health and Medical Subjects, No.* 78. H.M.S.O., London, 1935.

(57) Reynolds, E. S., 'An Account of the Epidemic Outbreak of Arsenical Poisoning Occurring in Beer Drinkers in the North of England and the Midland Counties in 1900'. *Lancet*, 1901, 1, 166.

(58) Annotation, 'Arsenic in the House'. *Lancet*, 1956, 2, 182.

(59) Gardner, C. A., and Bennetts, H. W., *Toxic Plants of Western Australia*. Perth, 1956, p. 150.

(60) British Plastics Federation, *Report of the 'Toxicity' Sub-Committee of the Main Technical Committee*. London, 1958.

(61) Min. Housing and Local Gov., *Report of the Committee on Synthetic Detergents*. H.M.S.O., London, 1956.

(62) Hueper, W. C., 'The Potential Role of Non-Nutritive Additives and Contaminants as Environmental Carcinogens'. *Unio Internat. Contra Cancrum, Acta*, 1957, 13, 220.

(63) U.S. Depart. Health, Education, Welfare, *Survey of Compounds which have been Tested for Carcinogenic Activity. Sup.* 1., Washington, 1957, p. 1 *et seqq.*

(64) Melnick, D. *et al*, 'Sorbic Acid as a Fungistatic Agent for Foods'. *Food Res.*, 1956, 21, 64.

(65) Burr, G. O. and Barnes, R. H., 'Non-Caloric Functions of Dietary Fats'. *Physiol. Rev.*, 1943, 23, 256.

(66) *Min. of Food, Food Standards Committee, Report on Antioxidants.* H.M.S.O., London, *1954.*

(67) Ottaway, F. J. H., and Coppock, J. B. M., 'Biscuit Stability. The Effect on Oxidative Changes of Certain

Natural and Synthetic Antioxidants'. *J. Sci. Food, Agric.*, 1958, 9, 294.

(68) Peacock, P. R., 'The Carcinogenic Action of Heated Fats and Lipoids'. *Brit. Med. Bull.*, 1947, 4, 364.

(69) Min. of Food, Food Standards Committee, *Report on Emulsifying and Stabilizing Agents*. H.M.S.O., London, 1956.

(70) Levin, R., *The Pharmacy of Silicones*. London, 1958, p. 4.

(71) Barnes, Ella M., 'New Methods in Food Preservation. Antibiotics'. *Roy. Soc. Health J.*, 1957, 77, 447.

(72) Love, R. M., 'Post-Mortem changes in the Lenses of Fish Eyes'. *J. Sci. Food, Agric.*, 1956, 7, 221.

(73) Hannan, R. S., *Scientific and Technological Problems Involved in Using Ionizing Radiations for the Preservation of Food*. H.M.S.O., London, 1955, p. 1 *et seqq*.

(74) Cutting, W. C., 'Toxicity of Silicones'. *Stanford Med. Bull.*, 1952, 10, 23.

(75) Oppenheimer, B. S. *et al*, 'Further Studies of Polymers as Carcinogenic Agents in Animals'. *Cancer Res.*, 1955, 15(1), 333.

(76) Deichmann, W. B. *et al*, 'Toxicity of Ditertiarybutylmethylphenol'. *Arch. Indust. Health*, 1955, 11, 93.

(77) Poling, C. E. *et al*, 'Effects of Feeding Polyoxyethylene Preparations to Rats and Hamsters'. *Food Research*, 1956, 21, 337.

(78) Todd, W. R. *et al*, 'On the Metabolism of Sorbitol and Mannitol'. *J. Biol. Chem.*, 1939, 127, 275.

(79) Payne, W. W. *et al*, 'Sorbitol (Sionon) for Diabetics'. *Lancet*, 1933, 2, 1257.

(80) Peters, R. and Lock, R. H., 'Laxative Effect of Sorbitol'. *Lancet*, 1958, 2, 677.

(81) Nelson, A. A. *et al*, 'Production of Bladder Stones

1956'. *Butterworth's Scientific Publications*. London, 1957, pp. 121, 129, 215.

(110) Barnes, J. N., *Toxic Hazards of Certain Pesticides to Man*. Geneva, 1953, pp. 53, 60, 63.

(111) Leading Article, 'Does BHC Damage the Bone Marrow?' *Brit. Med. J., 1958, 2, 1,522.*

(112) Roy, E. E. *et al*, 'Observations on Lindane Poisoning in Dairy Animals'. *J. Amer. Vet. Med. Ass.*, 1953, 123, 448.

(113) Laug, E. P., 'Occurrence of DDT in Human Fat and Milk'. *Arch. Indust. Hygiene*, 1951, 3, 245.

(114) Hayes, W. J. *et al*, 'The Effect of Known Repeated Oral Doses of Chlorophenothane (DDT) in Man'. *J. Amer. Med. Ass., 1956, 162, 890.*

(115) Kitselman, C. H., 'Long Term Studies on Dogs Fed Aldrin and Dieldrin in Sublethal Doses, with Reference to the Histopathological Findings and Reproduction'. *J. Amer. Vet. Med. Ass.*, 1953, 123, 28.

(116) Fitzhugh, O. G. and Nelson, A. A., 'The Chronic Oral Toxicity of DDT'. *J. Pharm., 1947, 89, 18.*

(117) Pool, W. A., 'Hyperkeratosis in Cattle'. *Medical Annual*, 1954, p. 441.

(118) Hansel, W. *et al*, 'The Isolation and Identification of the Causative Agent of Bovine Hyperkeratosis (X-Disease) from a Processed Wheat Concentrate'. *Cornell Vet.*, 1955, 45, 94.

(119) Spighel, A. and Alroy-Spighel, M., 'Metaldehyde Poisoning'. *Lancet, 1958, 2, 1,017.*

(120) Saunders, B. C., *Some Aspects of the Chemistry and Toxic Action of Organic Compounds Containing Phosphorus and Fluorine*. Cambridge, 1957, p. 170.

(121) Parkin, E. A., 'A Provisional Assessment of Malathion for Stored-Product Insect Control'. *J. Sci. Food Agric.*, 1958, 9, 370.

(122) Spoon, Invloed van systemische Insecticiden op de Cacao-Boon. *Voeding*, 1954, 15, 67.

(123) Bidstrup, P. L. *et al*, 'Paralysis Following Poisoning by a New Organic Phosphorus Insecticide (Mipafox)'. *Brit. Med. J.*, 1953, 1, 1,068.

(124) Barnes, J. M. and Denz, F. A., 'Experimental Demyelination with Organo-Phosphorus Compounds'. *J. Path. Bact.*, 1953, 65, 597.

(125) Hunter, D. *et al*, 'Toxic Polyneuritis Arising during the Manufacture of Tricresyl Phosphate'. *Brit. J. Indust. Med.*, 1944, 1, 227.

(126) Biskind, M. S., *On Certain Aspects of the Origins and Treatment of Communicable Disease, with Particular Reference to Viruses*. Westport, 1955, p. 4

(127) 'New Insecticide: Safe Use Nearer to Cropping Time', *The Times*, 1951, Feb. 8.

(128) 'A New Systemic Insecticide', *Nature*, 1951, 167, 260.

(129) 'A Pre-Harvest Insecticide: Beneficial Insects are not Harmed', *Farmers' Weekly*, 1951, Feb. 9.

(130) Advertisement, *Farmers' Weekly*, 1951, March 2 and 16.

(131) Koelle, G. B. and Gilman, A., 'The Chronic Toxicity of Di-Isopropyl Fluorophosphate (DFP) in Dogs, Monkeys, Rats'. *J. Pharm.*, 1946, 87, 435.

(132) Durham, W. F. *et al*, 'Paralytic and Related Effects of Certain Organic Phosphorus Compounds'. *Arch. Indust. Health*, 1956, 13, 326.

(133) Lehmann, H., 'Pseudocholinesterase'. *Lancet*, 1959, 1, 467.

(134) Wescoe, W. C. *et al*, 'Regeneration Rates of Serum Cholinesterase in Normal Individuals and in Patients with Liver Damage'. *Amer. J. Physiol.*, 1947, 149, 549.

(165) Sinclair, H. M., 'The Composition and Nutritive Value of flour'. *Roy. Soc. Health J.*, 1957, 77, 234.

(166) Report, *The Times*, 1958, Sept. 25.

(167) Edwards, J. H., 'Congenital Malformations of the Central Nervous System in Scotland'. *Brit. J. Prev. Soc. Med.*, 1958, 12, 115.

(168) Cantarow, A. and Trumper, M., *Lead Poisoning.* Baltimore, 1944.

(169) Pleydell, M. J., 'Epidemiological Studies of Huntington's Chorea and Congenital Abnormalities'. *Roy. Soc. Health J.*, 1958, 78, 599.

(170) Sinclair, J. G., 'A Specific Transplacental Effect of Urethane in Mice'. *Texas Rep. Biol. Med.*, 1950, 8, 623.

(171) Larsen, C. D., 'Pulmonary-Tumor Induction by Transplacental Exposure to Urethane'. *J. Nat. Cancer Inst.*, 1947, 8, 63.

(172) Dogramaci, I. and Green, H., 'Factors in the Etiology of Congenital Heart Anomalies'. *J. Pediat.*, 1947, 30, 295.

(173) Silbernagel, Wynne M. and Patterson, J. B., 'Wheat Germ Oil Concentrate as an Aid in the Reduction of Pregnancy Complications'. *Ohio State Med. J.*, 1951, 47, 533.

(174) Lindan, O. and Himsworth, H. P., 'Tocopherol and Protein Deficiency in Relation to the Development of Dietetic Massive Necrosis of the Liver in Rats'. *Brit. J. Exp. Path.*, 1950, 31, 651.

(175) Steyn, D. G., *The Problem of Dental Caries and the Fluoridation of Public Water Supplies.* Johannesburg, 1958, p. 32.

(176) Bransby, E. R. and Forrest, J. R., 'The Dental Condition of Children in Seven Areas in England and Wales'. *Mon. Bull. Min. Health*, 1958, 17, 28.

stitial Cell Tumours in Hybrid Mice Given Tri-*p*-Anisyl Chloroethylene'. *Arch. Path*, 1950, 50, 750.

(219) Pan, S. C. and Gardner, W. U., 'Carcinomas of the Uterine Cervix and Vagina in Estrogen and Androgen Treated Hybrid Mice'. *Cancer Res.*, 1948, 8, 337.

(220) Gardner, W. U. and Rygaard, J., 'Further Studies on the Incidence of Lymphomas in Mice Exposed to X-rays and Given Sex Hormones'. *Cancer Res.*, 1954, 14, 205.

(221) Min. Agric. Fish. Food, *Toxic Chemicals in Agriculture: Risks to Wild Life*. H.M.S.O., London, 1955, p. 28.

(222) Tayler, E. L., 'Adverse Effects on Animal Health Brought About by Man's Interference with Nature'. *Vet. Rec., 1955, 67, 1,036.*

(223) Papworth, D. S., 'A Review of the Dangers of Warfarin Poisoning to Animals other than Rodents'. *Roy. Soc. Health J.*, 1958, 78, 52.

(224) George, J. L., *The Pesticide Problem*. The Conservation Foundation, New York, 1957, pp. 17, 18, 20, 22.

(225) Kelley, C. D., 'Fire Ant and Fire Works'. *23rd Ann. Conven. Nat. Wildlife Fed.*, Washington, 1958, pp. 5–8.

(226) National Audubon Soc., *The Hazards of Broadcasting Toxic Pesticides*. New York, 1958, p. 3.

(227) Patuxent Research Refuge, *Abstracts of References on the Effects of Dieldrin on Wildlife*. Laurel, Maryland, U.S.A., 1958, p. 2.

(228) Keymer, I. F., 'A Survey and Review of the Causes of Mortality in British Birds and the Significance of Wild Birds as Disseminators of Disease'. *Vec. Rec.*, 1958, 70, 713.

(229) Jennings, A. R., 'Some Cases of Deaths in Wild Birds in Britain'. *Vet. Rec.*, 1958, 70, 758.

(230) 'Poison Found in Pigeons', *Farmers' Weekly*, 1959, May 22.

(231) 'Insecticides Upsetting Balance of Nature', *East African Standard*, 1959, May 26.

(232) Wallace, G. J., 'Insecticides and Birds'. *54th Ann. Conven. National Audubon Soc.*, New York, 1958, pp. 8, 13.

(233) Barker, R. J., 'Notes on some Ecological Effects of DDT sprayed on Elms'. *J. Wildlife Management*, 1958, 22, 269.

(234) Carnaghan, R. B. A. and Blaxland, J. D., 'The Toxic Effects of Certain Seed Dressings on Wild and Game Birds'. *Vet. Rec.*, 1957, 69, 324.

(235) Hall, C. B., *Ponds and Fish Culture for Pleasure or Profit*. London, 1949, pp. 19, 27.

(236) Berlin, R., 'Haff Disease in Sweden'. *Acta Med. Scandinav.*, 1947, 129, 560.

(237) Duncan, C., 'Discussion on River Pollution in Tidal Waters'. *Roy. Soc. Health J.*, 1958, 78, 770.

(238) Horne, A., 'Progress Threatens the Salmon'. *The Scotsman*, 1959, July 21.

(239) 'Thousands of Fish Poisoned', *Angling Times*, 1959, June 5.

(240) 'Crop Spray Blamed for Fish Deaths', *The Times*, 1958, Aug. 7.

(241) Burden, E. H. W. K., 'A Case of DDT Poisoning in Fish'. *Nature*, 1956, 178, 546.

(242) Tweed River Purification Board, *Fourth Annual Report*. Galashiels, 1958, pp. 6, 26.

(243) McAlpine, D and Araki, S., 'Minamata Disease. An Unusual Neurological Disease Caused by Contaminated Fish'. *Lancet*, 1958, 2, 629.

(244) Mathis, W. and Quarterman, K. D., 'Field Investigations on the Use of Heavy Doses of Several Chlorinated Hydrocarbons as Mosquito Larvicides'. *Amer. J. Trop. Med.*, 1953, 2, 318.

(245) Min. Housing and Local Government, *Second Progress Report of the Standing Technical Committee on Synthetic Detergents*. H.M.S.O., London, 1959, p. 7.

(246) Armitt, J. H. 'Pollination for Fruit and Seed'. *Brit. Bee J.*, 1956, March 29.

(247) Richards, F. A., 'Facts and Figures'. *Brit. Bee J.*, 1956, April 12.

(248) 'Beans, Bees and Yields', *Farmers' Weekly*, 1959, May 22.

(249) Barbier, E. C., 'Effect of Honey Bees on Lavender Oil Production'. *Brit. Bee J.*, 1959, Feb. 14.

(250) 'Harry', Warwickshire, 'Combings'. *Brit. Bee J.*, 1956, April 26.

(251) Cox, T. J., 'In the South'. *Brit. Bee J.*, 1956, March 1.

(252) Editorial, *Brit. Bee J.*, 1959, June 20.

(253) Tonsley, C. C., *Brit. Bee J.*, 1956, Jan. 12.

(254) Glynne Jones, D. G. and Edwards, R. A., 'Studies of Toxicity of 3:5-Dinitro-*ortho*-cresol and its Sodium Salt to the Honey Bee'. *Bull. Entomolog. Res.*, 1952, 43, 67.

(255) Thomas, W. D. E. and Glynne Jones, G. D., 'The Systemic Properties of Diethyl-S-2-(Ethylthioethyl) Phosphorothiolate (Demeton-S) with Reference to the Contamination of Nectar'. *Ann. Appld.* Biol., 1955, 43, 182.

(256) Thomas, W. D. E. and Glynne Jones, G. D., 'Experiments on the Possible Contamination of Honey with Schradan'. *Ann. Appld. Biol.*, 1953, 40, 546.

(257) Annotation, *Metal Industry*, 1957, 90, 62.

(258) Allen, H., 'Notes on Nectar Secretion'. *Brit. Bee J.*, 1957, Feb. 2 and Dec. 21.

(259) Sykes, F., *Humus and the Farmer*. London, 1946, p. 222.

(260) Hazleton, L. W. *et al*, 'Toxicity of Coumarin'. *J. Pharm. Exp. Therap.*, 1956, 118, 348.

(261) Coppock, J. B. M. and Knight, R. A., 'Polytetrafluoroethylene Films in Baking'. *Brit. Med. J.*, 1957, 2, 355.

(262) Brown, H. M. *et al*, 'Glycyrrhetinic Acid Hydrogen

Succinate (Disodium Salt). A New Anti-inflammatory Compound". *Lancet*, 1959, 2, 492.

(263) Annotation, *Time*, 1959, Nov. 2.

(264) Lish, P. M. and Weikel, J. H., 'Influence of Surfactants on Absorption from the Colon'. *Tox. Appl. Pharm.*, 1959, 1, 501.

(265) Zimmerman, M. C., 'Chronic Penicillin Urticaria from Dairy Products, Proved by Penicillinase Cures'. *Arch. Dermatol.*, 1959, 79, 1.

(266) Thewanger, W., 'Über den Nachweis einiger peroral verabreichter Antibiotika im Serum und in der Muttermilch der Wöchnerin'. *Klin. Med. Vienna*, 1958, 13, 486.

(267) Kitselman, C. H. *et al*, 'Toxicological Studies of Aldrin (Compound 118) on Large Animals'. *Amer. J. Vet. Res.*, 1950, 11, 378.

(268) Telford, H. S. and Guthrie, J. E., 'Transmission of the Toxicity of DDT through the Milk of White Rats and Goats'. *Science*, 1945, 102, 647.

(269) Wilson, R. H. *et al*, 'The Toxicity and Carcinogenic Activity of 2-Acetaminofluorene'. *Cancer Research*, 1941, 1, 595.

(270) Shier, F. L. and Rossiter, R. C., 'Clover Disease'. *J. Agric. West Australia*, 1949, 26, 111.

(271) Lancum, F. H., *Wild Mammals and the Land.* H.M.S.O., London, 1957, p. 1 et seqq.

(272) Lancum, F. H., *Wild Birds and the Land.* H.M.S.O. London, 1956, p. 1 et seqq.

(273) Satchell, J. E., 'Earthworm Biology and Soil Fertility'. *Soils and Fertilizers*, 1958, 21, 209.

(274) Darwin, C., *The Formation of Vegetable Mould through the Action of Worms, with Observations on their Habits.* London, 1882, p. 131.

(275) Beidas, A. H. and Higgons, D. J., 'Mercury Resi-

dues in Sprayed Crops: Apples'. *J. Sci. Food Agric.*, 1959, 10, 527.

(276) Smith, Honor and Spalding, Y. M. K., 'Outbreak of paralysis in Morocco due to Ortho-cresyl phosphate poisoning'. *Lancet*, 1959, 2, 1,019.

(277) Green, R. C., 'Nutmeg Poisoning'. *J. Amer. Med. Ass.*, 1959, 171, 1,342.

(278) Goldstein, N. P. *et al*, 'Peripheral Neuropathy after Exposure to an Ester of Dichlorophenoxyacetic Acid'. *J. Amer. Med. Ass.*, 1959, 171, 1,306.

(279) White, J. W. and Riethof, Mary L., 'Detection of Acetylandromedol in Toxic Honeys'. *Arch. Biochem. Biophys., 1959, 79, 165.*

(280) 'Arsenites in Agriculture', *Lancet*, 1960, 1, 178.

(281) Hall, L. P. and Raven, T. W., 'Spoilage of Canned Peas: A Thermophile that Affects the Artificial Colour'. *J. Sci. Food Agric.*, 1959, 10, 456.

(282) Sharman, I. M. and Richards, Pamela J., 'Commercial Breads as Sources of Vitamin E for Rats as Determined by the Haemolysis Test'. *Brit. J. Nutrit.*, 1960. 14, 85.

(283) Coleby, B. *et al*, 'Radiation Pasteurization of Whole Eviscerated Chicken Carcasses'. *J. Sci. Food Agric.*, 1960, 11, 61.

(284) Clements, F. W., 'Naturally Occurring Goitrogens'. *Brit. Med. Bull.*, 1960, 16, 133.

(285) Davies, D. R. *et al*, 'The Relationship between the Chemical Structure and Neurotoxicity of Alkyl Organophosphorus Compounds'. *Brit. J. Pharmacol.*, 1960, 15, 261.

(286) Davies, R. I. *et al*, 'Cancer Incidence in North Wales and Cheshire and in Two Devonshire Towns'. *Brit. Emp. Cancer Campaign, Scientific Rep.*, 1960, p. 365.

(287) Haddow, A. and Horning, E. S., 'On the Carcinogenicity of an Iron-Dextran Complex'. *J. Nat. Cancer. Instit.*, 1960, 24, 109.

(288) Carey, Fiona M. *et al*, 'Pharmacological and Chemical Observations on Some Toxic Nectars'. *J. Pharm.*, 1959, 11, 269.

(289) Wilson, G. S. *et al*, 'A Small Outbreak of Solanine Poisoning'. *Mon. Bull. Min. Hlth.*, 1959, 18, 207.

(290) Smith, H. W. and Crabb, W. E., 'The Effect of Diets Containing Tetracyclines and Penicillin on the *Staphylococcus Aureus* Flora of the Nose and Skin of Pigs and Chickens and their Human Attendants.' *J. Path. Bact.*, 1960, 79, 243.

(291) Annotation, *Farmers' Weekly*, 1960, June 3rd.